ENCYCLOPEDIA AND DICTIONARY

OF

MEDICINE AND SURGERY

Printed by R. & R. Clark, Limited, *Edinburgh*,

FOR

WILLIAM GREEN & SONS.

1907.

GREEN'S ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

VOL: IV

GUM-RESINS TO INTUSSUSCEPTION

WILLIAM GREEN & SONS
EDINBURGH & LONDON

SCOTT CALDER

EDITORIAL NOTE

This, the fourth, volume of the *Encyclopedia and Dictionary of Medicine and Surgery* carries the work from Gum-Resins to Intussusception, and contains 872 subject-headings. The four volumes now issued contain almost all the articles of the first five volumes of the *Encyclopædia Medica*, along with a great mass of new material, consisting for the most part of shorter contributions, but embracing also not a few comparatively long ones.

Of the 872 subject-headings in the present volume, 52 are those of articles of more than 1000 words in length, and one of these (that dealing with the Heart and its Diseases) occupies 98 pages, while another (that concerned with the various aspects of Insanity) covers 77 pages. New articles on *Diagnosis in Gynæcology,* on *Heart Block, Hirsuties, Homo Caudatus,* and on *Recent Views on Immunity* (including the *Opsonic Index*) have been added; and the contribution dealing with the *Pathology of Insanity* has been re-written. Other important articles in this group are those on *Hæmatemesis,* on *Hæmatoma Auris,* on *Hæmaturia,* on *Hæmophilia,* on *Hæmoptysis,* on *Hæmorrhage,* on *Hay Fever,* on *Headache,* on *Hemiplegia,* on *Heredity,* on *Hernia,* on *Herpes,* on the *Hip-Joint,* on *Hydatid Disease,* on *Hydrocephalus,* on *Hypnotism,* on *Hysteria,* on *Ichthyosis,* on *Indigestion,* on *Infant Feeding,* on *Infection,* on *Influenza,* and on the *Intestines.*

Of articles varying in length from 10 lines to 1000 words there are in this volume 54. These include paragraphs dealing with *Gums, Hæmatoma, Hæmatoxyli Lignum, Hæmochromatosis, Hæmoglobinometer, Hallux, Hamamelis, Head-Shaking, Surgery of the Heart, Hebotomy, Hegar's Sign, Hemeralopia, "Hemisine," Heroin, Hirudo, Hoffmann's Anodyne, Holocaine, Honey, Hook, Hops, Hyaline Degeneration, Hydramnios, Hydrastis, Hydrochloric Acid, Hydrocyanic Acid, Hydrogen Peroxide, Hydrops, Hydroquinone, Hydrorrhœa, Hyoscyamus, Hyperæsthesia, Hyperpyrexia, Hypnotics, Hypoglossal Nerve, Hypophosphites, Hypospadias, Hysterectomy, Ichthyol, Indian Drugs, Infancy, Infarction, Inhalations, Iniencephaly, Inoscopy,* and *Intubation.*

Of subject-headings which are of less than 10 lines in length there are 766. Many of these are short definitions of new terms, or short descriptions of new methods of treatment and of new drugs, while others are cross-references. A general idea of their nature will be gained from the enumeration of some of them, viz. *Gum-Resins, Gurjun Oil, Gut-Cleaning, Guy's Pill, Gynandry, Gyrus, Habit Spasm, Habitual Abortion, Habitus, Hæmalops, Hæmarthrosis, Hæmathidrosis, Hæmatocolpos, Hæmatocrit, Hæmatogenesis, Hæmatometra, Hæmatosalpinx, Hæmatotrachelos, Hæmocytolysis, Hæmogallol, Hæmokonia,*

Hæmolymph Glands, Hæmolysis, Hæmophagocytes, Hæmosiderin, Hæmostatics, Haffkine's Prophylactic, Hair, Halitus, Hammer Toe, Hamulus, Hanot's Disease, Hapalonychia, Haptophore Group, Hard Soap, Harrison's Groove, Haustus, Haut Mal, "Hazeline," Heart-burn, Heberden's Nodes, Hectic Fever, Hedonal, Hehner's Formula, Helalin, Helebore, Hellyer's Trap, Hemaboloids, Hemianopsia, Hemihypertrophy, Hemipagus, Hemiplégie Flasque, Hendon Epidemic, Henpuye, Hepatopexy, Herculesbad, Hermite Process, Hesselbach's Hernia and *Triangle, Heterochronism, Heterodymus, Heteropagus, Hetol, Hiatus, Himrod's "Cure," Hinckes-Bird's Method, Hippocratic Facies, Hippuria, Hippus, Hives, Hoffa's Disease, Homicide, Hominy, Horsley's Putty, Hospitalism, Hutchinson's Triad, Hyalophagia, Hydraulic Test, Hydromyelia, Hydroperione, Hyperacanthosis, Hyperbulia, Hyperepinephry, Hyperpyræmia, Hyperthyroidism, Hypodermoclysis, Hypoepinephry, Hypoxanthin, Hysterexopexy, Hystricismus, Ichor, Ichthalbin, Ictus Solis, Ignipuncture, Ikota, Immigration, Imperial Drink, Induration, Inertia, Infantilism, Inhalation-Pneumonia, Inosite, Inquisition, Insomnia, Intention-Tremor, Intimitis, Introitus,* etc., etc.

The cross-references have, as in the earlier volumes, been prepared with great care, and will, it is hoped, prove of much value in facilitating the employment of the work by the busy practitioner.

J. W. BALLANTYNE.

April 13, 1907.

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Gum - Resins. — Ammoniacum, Cambogia, Galbanum, and Scammonium are official gum-resins; they are exudations from plants, and consist of gums and resins mixed together. Since the gum is soluble, one can finely powder and rub the gum-resin in a mortar with a little water in order to make an emulsion (the resin being insoluble).

Gums.—(1) Gums are also exudations from plants, containing soluble or insoluble substances, such as *arabin* (in gum acacia) or *cerasin* (calcium metagummate or cherry-tree gum). *See* Gum Acacia; Gummi Indicum; etc. (2) The gums are the fleshy substance surrounding the jaws and teeth. *See* Children, Clinical Examination of (*Mouth, Gums*); Children, Development of (*Dentition, Lancing the Gums*); Gingivitis; *Malaria* (*Sequelæ, Bleeding from the Gums*); Menstruation and its Disorders (*Vicarious*); Mouth, Diseases of the (*Diseases of the Gums*); Scurvy, Infantile (*Clinical Features*); Scurvy in Adults (*Clinical Features*); Toxicology (*Plumbism, Symptoms, "Blue Line"*); Trades, Dangerous (*Lead-Poisoning, Symptomatology*).

Gunjah. *See* Cannabis Indica.

Gunshot Wounds. *See* Ankle-Joint, Region of, Injuries (*Wounds, Gunshot*); Knee-Joint, Injuries of (*Gunshot Wounds*); Medicine, Forensic (*Wounds from Fire-arms*); Œsophagus (*Wounds of Neck*).

Gurgling. *See* Typhoid Fever (*Symptoms, Gurgling in the Iliac Fossa*).

Gurjun Oil.—Gurjun oil or balsam or wood-oil is obtained from various species of Dipterocarpus by incision of the trunk of the tree; it resembles copaiba; and it has been used in gonorrhœa and occasionally in leprosy in doses of 10 to 30 m. *See* Leprosy (*Treatment*).

Gurnigel. *See* Balneology (*Switzerland, Canton Bern*).

Gusanopeludo. — The Dermatobia noxialis, which is believed to cause tropical cutaneous myiasis in the human subject.

Gustafsberg. *See* Balneology (*Sweden*).

Gut-Cleaning.—Gut-cleaning and gut-spinning for the purpose of making catgut, sausage skins, fiddle-strings, etc., are very offensive trades; and there are strict laws for their regulation (English Public Health Act, 1875; Scottish Act, 1897).

"Guthrie's" Hernia. *See* Hernia (*Causation, Blows*).

Gutta.—(1) A drop or a fluid preparation to be dropped into the eye; (2) gout; (3) an old term for apoplexy; and (4) a contracted form for guttapercha.

Gutta Percha.—The concrete "drop" or juice of the percha tree, of Isonandra gutta, and of other Sapotaceous plants; the Liquor Gutta Percha (not now official) was a solution of gutta percha in chloroform. Gutta percha is used for making splints, as a protective covering of the skin, for stopping tooth-cavities, etc.

Gutta Rosea. — Acne rosacea. *See* Acne (*Acne Rosacea.*)

Gutturals. *See* Physiology, Respiration (*Voice, Articulate Speech*).

Guy's Pill.—A diuretic pill containing mercury, digitalis, and squill (1 grain each of blue dill, squill root, and digitalis leaves, with $1\frac{2}{3}$ grain of extract of hyoscyamus).

Gymnastic Exercises. *See* Massage.

Gynæco-.—In compound words *gynæco-* signifies relating to women; thus, *gynæcology* (*q.v.*) means the study of the diseases peculiar to women, *gynæcomastia* means an enlargement of the mammary gland in the male, causing it to resemble the same organ in the female both in appearance and in the function of secreting milk (*see* MAMMARY GLAND, DISEASES OF, *Male Breast*), and *gynæcophobia* means a morbid dread or dislike of women.

Gynæcology. *See* ABDOMEN, CLINICAL EXAMINATION OF; ABDOMINAL TUMOURS, DIAGNOSIS OF; BLADDER, INJURIES AND DISEASES (*Female Bladder*); BROAD LIGAMENT, DISEASES OF; COCCYGODYNIA; CURETTAGE, UTERINE; CYSTOSCOPE; ECTOPIC GESTATION; FALLOPIAN TUBES; GENERATION, FEMALE ORGANS OF; GENERATION, FEMALE ORGANS OF, ARRESTED DEVELOPMENT; GONORRHŒAL INFECTION; GYNÆCOLOGY, DIAGNOSIS IN; HERMAPHRODITISM; HYSTERIA; MAMMARY GLAND, DISEASES OF; MENOPAUSE; MENSTRUATION AND ITS DISORDERS; NEURASTHENIA; OVARIES, DISEASES OF THE; PELVIS, PERINEUM AND PELVIC FLOOR; PELVIS, DISEASES OF THE PERITONEUM AND CELLULAR TISSUE; PERITONEUM; PUBERTY; PUDENDA, GRANULOMA OF; STERILITY; SYPHILIS; TUMOURS (*Myomata, Phantom Tumours*, etc.); URACHUS; URETHRA; UTERUS, MALFORMATIONS OF; UTERUS, DISPLACEMENTS OF; UTERUS, INFLAMMATIONS OF; UTERUS, NON-MALIGNANT TUMOURS OF; UTERUS, MALIGNANT TUMOURS OF; VAGINA, DISORDERS OF; VISCERAL PAIN; VULVA, DISEASES OF.

Gynæcology, Diagnosis in.

GENERAL PRINCIPLES: CASE-TAKING SCHEME

FOR the orderly arrangement of the facts elicited by the examination of a gynæcological case it is necessary to have a scheme or syllabus: that given below is the one in use in the Royal Infirmary, Edinburgh:—

A. **Anamnesis (Symptomatology)**—

1. NAME; AGE; OCCUPATION; RESIDENCE; CONDITION AS TO MARRIAGE (Single, Married, Widowed); DATE OF ADMISSION (or examination).
2. COMPLAINT AND DURATION OF ILLNESS.
3. GENERAL HISTORY OF—
   (*a*) Present Attack. (*b*) Previous Health. (*c*) Diathesis. (*d*) Social Condition and Habits. (*e*) Family Health.
4. SEXUAL HISTORY—
   (1) *Menstruation.*
   A. Normal—
   (*a*) Date of commencement (Menarché).
   (*b*) Type.
   (*c*) and (*d*) Duration and Quantity, or Habit.
   (*e*) Date of disappearance (Menopause).
   B. Morbid—
   (*a*) Amenorrhœa or Oligomenorrhœa.
   (*b*) Menorrhagia.
   (*c*) Dysmenorrhœa.
   (2) *Intermenstrual Discharge*—
   (*a*) Character. (*b*) Quantity.
   (3) *Pareunia.*
   (4) *Pregnancies*—
   (*a*) Number. (*b*) Dates of first and last. (*c*) Abortions. (*d*) Character of Labours. (*e*) Puerperia. (*f*) Lactations.
5. LOCAL FUNCTIONAL DISTURBANCES—
   (*a*) Bladder. (*b*) Rectum.
   (*c*) Pelvic nerves and muscles.
6. GENERAL FUNCTIONAL DERANGEMENTS—
   (*a*) Nervous system. (*b*) Respiratory system. (*c*) Circulatory system. (*d*) Digestive system.
   (*e*) Urinary system.

B. **Physical Examination**—

1. GENERAL APPEARANCE AND CONFIGURATION.
2. MAMMÆ.
3. ABDOMEN—
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   (*e*) Mensuration.
4. EXTERNAL PUDENDA.
5. PER VAGINAM—
   (*a*) Orifice. (*b*) Walls and Cavity. (*c*) Os and Cervix Uteri. (*d*) Roof or Fornices.
6. BIMANUAL EXAMINATION (Abdomino-Vaginal, Recto-Vaginal, Abdomino-Rectal, Abdomino-Recto-Vaginal).
   (1) *Uterus*—
   (*a*) Size. (*b*) Shape. (*c*) Consistence. (*d*) Sensitiveness. (*e*) Position. (*f*) Mobility.
   (*g*) Relations.
   (2) *Fallopian Tubes.*
   (3) *Ovaries*—
   (*a*) Size. (*b*) Situation.
   (*c*) Sensitiveness.
   (4) *Peritoneum and Cellular Tissue.*
   (5) *Bladder.*
   (6) *Rectum.*
   (7) *Pelvic Bones.*

7. USE OF—

(*a*) Speculum.	(*d*) Curette.
(*b*) Volsella.	(*e*) Aspiratory Needle.
(*c*) Sound.	(*f*) Dilator or Tent.

8. PHYSICAL CHANGES IN—

(*a*) Nervous system.	(*d*) Digestive system.
(*b*) Respiratory system.	(*e*) Urinary system.
(*c*) Circulatory system.	(*f*) Cutaneous system.

(*g*) Locomotory system.

C. **Diagnosis of Case.**
D. **Prognosis of Case.**
E. **Treatment.**
F. **Progress and Termination.**

SYMPTOMATOLOGY OF GYNÆCOLOGY (ANAMNESIS)

This part of the examination is conducted by questioning the patient (or her mother, if the patient be an unmarried girl) as to her symptoms; tact is required, for it is necessary to elicit all the facts without offending the patient's modesty or causing her to mislead us either intentionally or unwittingly. The information gained by the anamnesis is not of the same high value as that got from the physical examination (in competent hands), but it is not to be neglected.

1. PRELIMINARY INQUIRIES: (*a*) *name*, for purposes of reference, and in order at once to get idea of patient's state as regards marriage; (*b*) *age*, in order (i.) to be alert as to detection of diseases which are common at special periods of life, such as nervous affections at the menopause, cancer of the uterus about the same age, and myomata a little earlier, and (ii.) to help in deciding what kind of treatment is to be adopted; (*c*) *occupation*, in order to learn if any causal conditions exist, such as lifting heavy weights, long hours of standing, luxurious habits, etc.; (*d*) *residence*, for future reference, especially when it is desirable to trace the result of treatment; former residence in a malarious or tropical climate should also be noted; (*e*) *condition as to marriage*, keeping in mind that a woman may be single, married, or widowed, that she may have been twice married, and that she may be pregnant although unmarried or a widow; (*f*) *date of admission* (in hospital practice) or *examination* (in private practice).

2. COMPLAINT AND DURATION OF ILLNESS: (*a*) *complaint*, to be recorded in patient's own words, *e.g.* a "falling of the womb," "flooding," "white discharge," "pain in the back," etc.; and the gynæcologist will do well to familiarise himself with the popular terminology in use for the description of gynæcological complaints in the part of the country where he is settled; (*b*) *duration of illness* to be noted as exactly as possible, useful time-marks in the patient's history being the birthdays of her children; and from this information one may judge as to acute or chronic nature of malady.

3. GENERAL HISTORY: (A) PRESENT ATTACK OR ILLNESS, as to (i.) date of commencement, (ii.) first symptoms, (iii.) mode of advance, (iv.) development of fresh symptoms, (v.) treatment adopted, and (vi.) effects of treatment. Do not ask too many leading questions, but endeavour to prevent patient wandering from the point and giving much irrelevant information.

(B) PREVIOUS HEALTH, more particularly as to (i.) occurrence of similar attacks, and (ii.) the history of syphilitic or gonorrhœal infection (very carefully elicited, and *not* by direct interrogation).

(C) DIATHESIS may be noted (*e.g.* when a distinctly nervous, rheumatic, or gouty constitution is present), but is a somewhat minor detail.

(D) SOCIAL CONDITION AND HABITS must be carefully inquired into, and (the latter) with delicacy and tact, special attention being paid to want of exercise, indulgence in alcohol or drug-taking (*e.g.* for painful menstruation), and tea-drinking in excess.

(E) FAMILY HEALTH, especially in relation to occurrence of cancer (call it "tumours"), of phthisis (call it "lung troubles"), or difficult labours, etc., in near relatives.

4. SEXUAL HISTORY is most important in a gynæcological case.

(1) MENSTRUATION: if (*a*) *normal*, inquire into its date of commencement (menarché), its type (cyclical recurrence), its habit (duration and quantity), and (in elderly women) its date of disappearance (menopause); and the reader may here usefully read the article dealing with MENSTRUATION AND ITS DISORDERS; if (*b*) *morbid or abnormal*, inquire (*a*) into the form of the abnormality, *e.g.* (i.) amenorrhœa, or absence of menstruation, including oligomenorrhœa (scanty menstruation) and cryptomenorrhœa (concealed or retained menstruation); (ii.) menorrhagia, or excessive menstruation; and (iii.) dysmenorrhœa, or painful menstruation; and (*b*) into the degree of the abnormality (*e.g.* amount of excess, presence of clots, character of the pain, etc.). (*Note.*—The physical examination will be necessary in order to discover the cause of the abnormality.)

(2) INTERMENSTRUAL DISCHARGE OR PAIN: there may be (*a*) metrorrhagia, or a red (blood) discharge in the intermenstrual period; or (*b*) leucorrhœa, a white (or yellow) discharge, which may come from the vulva or vagina (acid in reaction), or from the uterine cervix or body (alkaline in reaction), and may contain various histological elements, according to its origin; or (*c*) Mittelschmerz, or Mid-Pain, a pain occurring at the mid time between two menstruations.

(3) PAREUNIA: the occurrence of pain during coitus (dyspareunia) is occasionally a symptom which it is of importance to elicit; but usually no inquiries need be made under this heading.

(4) PREGNANCIES: inquire (*a*) as to number of pregnancies the patient has had, including those that ended prematurely as abortions; (*b*) the dates of the first and last, often ascertained

most easily by asking the ages of the youngest and of the oldest child; (*c*) the number of abortions, the age of the pregnancy (*e.g.* third month) when they occurred, and whether medical attention was then sought for and obtained; (*d*) the character of the labours, normal, laborious, preternatural, or complex; (*e*) the course, normal or abnormal, of the puerperal periods, especially in regard to the occurrence of fever or hæmorrhage in the days following labour; and (*f*) the duration and course of the lactations or nursing periods, and the occurrence of mammary troubles.

5. Local Functional Disturbances.—(1) Bladder: inquire whether there is pain during or after passing water (dysuria), or retention (inability to pass water), or incontinence (loss of power of holding water), for such symptoms may point to uterine disorders (*e.g.* displacements) or to fistulæ (*e.g.* vesico-vaginal).

(2) Rectum: inquire into the existence of habitual or occasional constipation, or of diarrhœa, or of pain on defæcation (emptying of the bowel), for these symptoms may be associated either as causes or effects of uterine and ovarian troubles.

(3) Pelvic Nerves and Muscles: sometimes pelvic pains (sacralgia), or pains shooting down the thighs, are associated with gynæcological diseases (*e.g.* cancer, cellulitis); sometimes, also, muscular weakness occurs (*e.g.* in rupture of the perineum) or muscular spasm (*e.g.* in vaginismus).

6. General Functional Derangements: although the attention of the practitioner is specially directed towards the symptoms localised in the pelvic organs, he must not forget to inquire into the occurrence of symptoms in the more distant parts of the body (*e.g.* in the nervous, respiratory, circulatory, digestive, and urinary systems); for they may have a connection with the gynæcological complaint, either the general symptom being a consequence of the pelvic trouble, or (less commonly) the pelvic symptom indicating a general disease; thus, dyspnœa may be a pressure symptom due to a large ovarian cyst, palpitation may be caused by metrorrhagia in uterine fibroids, and pain in a distant part (*e.g.* the heel) have its origin in a urethral caruncle, while dysuria may be the result of renal disease, and pain in the back indicate lumbago of rheumatic origin.

Physical Examination in Gynæcology

1. General Appearance and Configuration to be noted when the patient walks into the room and during the anamnesis, *e.g.* anæmic or cachectic appearance, abdominal enlargement, crookback, dwarfism, etc.

2. Mammary Glands: these are examined by inspection and palpation, in order, early in the investigation, to get a hint of the existence of pregnancy, for, in dealing with a patient supposed to be gynæcological, this possibility must be kept in mind. "Ovarian tumours" have sometimes turned out to be pregnancies, and *vice versa*; therefore, be alert to detect any or all of the seven mammary signs of pregnancy. In other cases the breasts are examined to detect mammary abscess, cyst, cancer, or fissure of nipple, and to determine their capacity for lactation.

3. Abdomen.—(1) Inspection: (*a*) *Method*: position of patient, dorsal, on consulting-room couch, or in bed; her head supported on a pillow; the bladder and rectum should be empty; her clothes are drawn up to her waist under the covering, which is then folded down over the examiner's right hand, placed at the level of the symphysis pubis, leaving the abdomen but not the general genitals exposed; (*b*) *Details*: note state of skin (pigmentation, etc.) and cutaneous veins; condition of distension, local or general; and umbilicus (depressed, flattened out, herniated); (*c*) *Description*: in recording what is observed, make use of the regional division of the abdomen into nine areas by two horizontal lines (upper one joining tenth costal cartilages at their most prominent parts, lower one uniting the two iliac crests where most prominent), and two vertical ones (from eighth costal cartilage to mid point in Poupart's ligament on each side); for, in this way, swellings and other irregularities can be localised in one or other or several of these areas. (*Note.*—The reader should here recall to mind the anatomical contents of each region.)

Right Hypochondriac	Epigastric	Left Hypochondriac
Right Lumbar	Umbilical	Left Lumbar
Right Iliac	Hypogastric	Left Iliac

(2) Palpation: (*a*) *Method*: with warmed *hands* (wash in hot water, which both warms and increases tactile sense of skin) palpate firmly and deeply, beginning with the lower zone of regions (iliacs and hypogastric); patient should lie easily and comfortably, with her knees drawn up, and should be engaged in conversation, so as to relax the abdominal muscles; (*b*) *Details*: note feeling and thickness of skin and subcutaneous tissue; sensation of resistance or tenderness or fluctuation at any part; size, shape, and consistence of any swelling found either in the abdominal walls or cavity, and its connection with the pelvic structures (*Note.*—The hand cannot be insinuated at the pelvic brim below tumours which have grown up out of the pelvis) and other organs; presence of swellings (*e.g.* enlarged glands or herniæ) in the inguinal regions, as indicative of possible venereal mischief or inflammation of vulva and lower part of vagina.

(3) Percussion: (*a*) *Method*: make deep

percussion, as a rule, first with patient on the back and then lying on the side and sitting up; (*b*) *Details*: from the percussion note we can often determine more exactly the outlines and nature of abdominal swellings, their relations, and the presence or absence of free fluid.

(4) Auscultation: (*a*) *Method*: with stethoscope or phonendoscope, usually in the hypogastric and iliac areas; (*b*) *Details*: the results are generally negative (*e.g.* no fœtal heart sounds), but may hear uterine souffle (in myomata), and, of course, pregnancy may be present although the case has been hitherto regarded as non-obstetrical.

(5) Mensuration: (*a*) *Method*: tape measure or cyrtometer; (*b*) *Details* may be useful in estimating rate of growth of abdominal enlargement.

4. Inspection of Pudenda: only necessary if there is suspicion of gonorrhœa or syphilis, when something is said "to come down" either in front (*e.g.* prolapsus uteri) or behind (*e.g.* piles), or if there is local tenderness (*e.g.* Bartholian abscess, or urethral caruncle); do not, in other cases, make this a routine practice.

5. Examination per Vaginam.—(1) Indications: (*a*) in all women who are *married* and who complain of symptoms pointing (even indistinctly) to a pelvic cause; if refused, then (save in the case of very nervous women seen for the first time and in whom the symptoms are not urgent) the gynæcologist should not consent to deal with the case; (*b*) in *unmarried* women, in whom the pelvic symptoms are neither urgent nor long continued, it is well to try first general or medicinal means of treatment, but if these fail a vaginal examination should be no longer postponed, *e.g.* in (α) menorrhagia, (β) amenorrhœa, and (γ) dysmenorrhœa which have not been benefited by such means as (α) regulation of the bowels, (β) iron and arsenic, and (γ) such a simple remedy (in ʒi. doses) as spiritus chloroformi and spiritus ammoniæ aromatici (āā. ℥ss.) made up with liquor ammoniæ acetatis (℥i.); the value of the rectal examination in young unmarried women is undoubted, but it is not free from objection.

Note.—The word examination, if unqualified by "of the chest" or "of the lungs" is understood by women to mean "the vaginal"; the presence of menstruation, save in emergencies, contra-indicates the vaginal examination.

(2) Method: the patient lies on her left side, with her hips near the edge of the couch or bed, and with the knees drawn up towards the abdomen; the gynæcologist thoroughly washes his hands with soap and water (using the nail brush), and anoints the fore and middle fingers of the right hand with vaseline (to facilitate entrance and give sensitiveness); he then folds these two fingers up in the palm and passes his hand towards the posterior part of the vulva, separating and keeping out of the way the patient's garments or the bedclothes with his left hand as he does so; having reached the perineum he extends the fore and middle fingers of the right hand and introduces them into the vaginal orifice which lies immediately in front. (*Note.*—It is well to practise the use of the left hand, for the position of the patient or the couch may make it necessary for the examination to take place with the woman lying on her right side.)

(3) Details to be noted: observe in order the following points:—

(*a*) Condition of the *vaginal orifice*: whether it be patulous or narrow, the state of the perineum (intact, ruptured, and to what extent), presence of growths attached to labia, protrusion of masses through vulva, pain or painful spasm (vaginismus) at orifice.

(*b*) Condition of the *walls and cavity*: size and shape of canal, presence or absence of rugæ on walls, temperature and moisture, tumours attached to walls or lying in canal, foreign bodies (plugs, pessaries), discharge (secretion), and character.

(*c*) Condition of *cervix uteri and os*: form, size, consistence, position, mobility, and direction of cervix; form, size, and direction of os; state of lips of os (fissured, lacerated, swollen); projection of tumours, fragments of abortion sac, or pessaries through os.

(*d*) Condition of *posterior vaginal fornix or fossa* (keeping in mind anatomical relations): presence of any swelling in it which causes it to project into the vaginal canal; such a swelling may be (α) fæces in rectum, tumours, (β) cellulitic (inflammatory) deposit round rectum, (γ) inflammatory deposit or effusion or ascitic fluid in pouch of Douglas, (δ) body of retroverted or retroflexed uterus (moving with cervix), (ε) tumour (fibroid) attached to posterior wall of uterus, (ζ) prolapsed or enlarged ovary or Fallopian tube (*e.g.* tubal pregnancy) in pouch of Douglas, (η) abdominal organs (*e.g.* kidney) or tumours growing from them which have gravitated into pouch of Douglas.

(*e*) Condition of *anterior fornix* (keeping in mind anatomical relations): a firm body felt through it may be body of uterus with or without tumour attached, cellulitic deposit, tumour or stone in bladder, or displaced ovary or tube.

(*f*) Condition of *lateral fornices* (keeping in mind anatomical relations): a swelling felt at side may be cellulitic deposit in broad ligament, extra-uterine pregnancy, ovarian or tubal growth, or hæmatoma; posteriorly a tense or contracted utero-sacral ligament may be felt. (*Note.*—Morbid states in the lateral fornices are usually associated with uterine displacement.)

6. Bimanual Examination: most important; the per vaginam is simply the first stage of the bimanual.

(1) Indications are the same as those for

the vaginal examination (*q.v.* antea); contra-indications are acute inflammation and advanced cancer of uterus.

(2) Method and Details: (*a*) the patient assumes the dorsal posture in an easy attitude (head comfortably supported, thighs flexed on the abdomen and abducted and rotated slightly outwards, and heels resting on the couch or bed); (*b*) the gynæcologist keeps the fore and middle fingers of the right hand in the vagina (as during the per vaginam) and places them in contact with the vaginal portion of the cervix with the palmar surface turned forwards; the thumb lies over the left labium majus and the little and ring fingers are folded in the palm of the hand; he then places the tips of the fingers of the left (or "abdominal") hand upon the anterior abdominal wall in the middle line about two inches above the symphysis pubis; the actual palpation can now be begun; (*c*) with the internal hand the perineum is pressed backward and the cervix is pushed upward, so that the fingers touching it lie in the axis of the pelvic brim; the fingers of the external hand then press downwards into the brim, and if the uterus is lying to the front, the fingers in the vagina feel an impact against them from the cervix, and this demonstrates that the uterus is in its usual position and is between the two examining hands; the learner may now reverse the procedure and press with the internal fingers sharply upwards, when he ought to feel the body of the uterus roll under his external hand placed on the abdomen; when the uterus is displaced backwards or much to the side, the fingers of the examiner's two hands will feel each other through the intermediate tissues; (*d*) the process above described is now repeated for the other parts and structures, the fingers palpating the sides and the back of the pelvis so as to note the size, consistence, form, and relations of the organs, growths, deposits, etc., lying there; (*e*) skill in this method of examining is only attained by constant practice, but when gained it is invaluable; (*f*) it is necessary for the bladder and rectum to be empty before a satisfactory bimanual examination can be made; and it is well to take in order all the organs and structures in the pelvis and examine each in turn (do not be satisfied with the detection of one lesion, *e.g.* retroversion of uterus); (*g*) a typically easy case is one in which the abdominal walls are thin and relaxed, the vagina roomy, the uterus slightly enlarged (*e.g.* a multiparous woman some days after labour), while a typically difficult one is found when the abdominal walls are thick and rigid, the vagina short and narrow, and the uterus poorly developed (*e.g.* a nervous nulliparous woman with an infantile uterus); (*h*) the *rationale* of the bimanual is found in the displacement of the segments of the pelvic floor (the posterior being driven backwards and the anterior upwards) and in the ease with which the uterus can be pressed against the anterior segment by the external hand.

(3) Varieties: (*a*) the form of bimanual above described is the commonest, and may be named abdomino-vaginal; but (*b*) the recto-abdominal (middle finger of right hand in the rectum, and left hand over abdomen) may be used, especially for the detection of structures lying behind the uterus, or in recto-vaginal septum; or (*c*) the recto-vagino-abdominal (middle finger of right hand in rectum, fore-finger in vagina, and left hand over abdomen) may be employed under similar circumstances, and in order to get a very thorough knowledge of the pelvic contents.

(4) Aids in performing the Bimanual: (*a*) the use of an *anæsthetic*, (*b*) the presence of the *sound* in the uterus, (*c*) the downward traction of the uterus by a *volsella*, and (*d*) the presence of a *sound* in the *bladder* will be of service in difficult cases.

6*a*. Rectal Examination: an occasional procedure in Gynæcology.

(1) Indications: (*a*) when the conditions found by the bimanual examination do not adequately account for patient's symptoms and state; (*b*) when there are symptoms pointing directly to anal or rectal disease; (*c*) when the patient is an unmarried girl or woman, and the vaginal touch is contra-indicated; (*d*) when a body lying posterior to the uterus has been detected per vaginam but not identified; and (*e*) when the vaginal touch is impossible on account of atresia, stenosis, or vaginismus, or from the presence of a tumour.

(2) Method: (*a*) before removing the fingers from the vagina the anterior rectal wall may be exposed by everting it through the anus; this is done by hooking the two "vaginal" fingers backward towards the coccyx and then pressing them outward towards the anus (Storer's method); in this way the rectal mucosa may be inspected and the use of a rectal speculum rendered unnecessary; (*b*) more commonly rectal "touch" is performed; the patient lies on the side or back; the gynæcologist washes his hands and draws the tip of his middle or index finger sharply across a cake of soap (in this way the crevice below the nail is filled with soap, which prevents fæcal matter lodging there, while the soap can easily be removed after the examination); the finger is then covered well with vaseline and introduced slowly and with a semi-rotatory movement into the anus, and directed at first forwards and then backwards and upwards; (*c*) rarely, the patient is put in the genu-pectoral position, and the inspection of the rectum (distended with air) is carried out with the help of a proctoscope, or (rarely) a sigmoidoscope, and an electric light (on the examiner's forehead), as practised by Howard Kelly; (*d*) the emptying of the rectum by

medicine or an enema is a necessary preliminary to these methods of examination.

(3) Details to be noted: in practising rectal touch, note (*a*) the presence of piles (external or internal), fissures, polypi, or strictures in the canal; (*b*) the direction, mobility, and relations of the coccyx, and its tenderness (coccygodynia); (*c*) the state of the cervix and body of the uterus as felt through the anterior rectal wall; (*d*) the presence and nature of bodies behind, above, or at the sides of the uterus (only possible if the uterus be depressed by the left hand over the abdomen, or drawn down by a volsella in the vagina attached to the cervix).

7. Examination aided by the Use of Instruments:—

(I.) *Vaginal Speculum.*

(1) Spatular Variety of Vaginal Speculum: this variety, including the well-known Sims' speculum, is the most useful, and the only truly scientific type.

(*a*) *Description*: Sims' speculum is really a double perineal retractor (in an emergency a spoon handle bent at an angle will serve the purpose), and consists of a handle with a "duck-bill" blade at each end at right angles to the handle; each blade is concave anteriorly and the one is larger than the other; the whole instrument is made of metal. There are many modifications; in one (Bozeman's) the blades are attached to the handle at a slightly acute angle, and in another (Simon's) there are several blades of different sizes and shapes, which can all be fixed to the same handle.

(*b*) *Method of Use*: (1) the *position* of the patient is the most important factor in the successful use of spatular specula: she may be placed in the genu-pectoral position, but since this is irksome, the semi-prone or Sims' position is that commonly employed for purely diagnostic purposes:[1] she lies on her left side and partly on her face on the couch, with the left arm drawn out from under her and hanging over the edge of the couch next to the gynæcologist, and with her thighs flexed upon the abdomen, the right one being drawn up farther than the left so as to touch, with its inner aspect, the couch above it; in this posture the pelvis is higher than the abdomen, and, therefore, when the lips of the vulva are separated and the perineum retracted, air rushes into the vagina through the displacement diaphragmwards of the anterior segment of the pelvic floor; (2) *Mode of Application*: the blade to be used is now chosen, and the instrument is washed, warmed, and vaseline is applied to the convex surface of the blade chosen; the other blade is then grasped in the left hand while two fingers of the right hand separate the labia; the blade is carried into the vagina and pushed onwards, following the curve of the posterior vaginal wall till it rests in the posterior fornix; if the perineum is now retracted, and the tip of the blade tilted forward, the vagina balloons and a good view is obtained of the cervix, the fornices, and the walls; if any further manipulation is intended, it is almost essential to have an assistant (not necessarily a trained one) to hold the speculum in position (Auvard's self-retaining duck-bill speculum, with its handle weighted with lead, can only be used in the lithotomy position).

[1] In operative work it is common to use the spatular speculum, with the patient in the lithotomy position.

(*c*) *Advantages and Disadvantage*: the great advantage of the spatular over the other forms of speculum is that, by utilising the separation of the segments of the pelvic floor produced by the effect of posture, it converts a closed canal into an open cavity, and so gives a better view of all the internal parts. Apart from diagnosis, it makes possible a long series of minor and major gynæcological operations on the uterus and vagina; its sole disadvantage is the need for an assistant to hold it in position if the gynæcologist contemplates operative interference or further diagnostic manipulations (*e.g.* use of uterine sound).

(*d*) *Cautions*: use gentleness; avoid traction on vulvar hairs; obtain a good light (daylight or portable electric light, *e.g.* on forehead); and precede use of speculum by per vaginal and bimanual examinations.

(*e*) *Contra-indications*: menstruation, pregnancy (as a rule), acute inflammation, cancer of cervix, and virginal state (as a rule).

(2) Tubular Variety of Vaginal Speculum: this variety, represented by the so-called Fergusson speculum, is now only used for diagnostic purposes by the general practitioner.

(*a*) *Description*: is tubular in shape, with a proximal trumpet-shaped end (to be held in the hand), and a distal bevelled end, in which the cervix lies (when the instrument is in position); the bevelling gives the tube a short (anterior) side, and a longer (posterior) one to suit the shape of the vagina; the maker's name (Fergusson) is often found at the proximal end of the anterior side; three sizes are common, measuring from a half to two inches in diameter, and they fit into each other, forming a "nest," but they are generally made too long for the normal vagina; the material is glass, coated with quicksilver, and covered with vulcanite or caoutchouc, and varnished; but they may be made of metal, ivory, porcelain, or celluloid.

(*b*) *Mode of Use*: (1) the patient lies on the left side, with hips near edge of couch and raised; (2) warm the speculum, and inspect it to see that there is no crack in the glass; put vaseline on its outer aspect; with the left hand raise the patient's right buttock and separate the labia with the fingers; hold the proximal end of the speculum in the right hand (thumb and three fingers) and introduce the distal end

into the vaginal orifice, keeping it well back (sensitive parts are in front), and push it onwards and upwards, keeping the short side to the front (guided by maker's name) until it meet with resistance; then look through it, and may find cervix is engaged in the end of it, but if not, withdraw it slightly and reintroduce till get view of cervix; with cotton wool on a sound remove mucus from cervix, so as to see it better.

(*c*) *Advantages and Disadvantages*: its advantages are that it can be easily introduced, involves slight exposure, is self-retaining, and does not necessitate the assumption of the Sims' position; but its disadvantages are that it gives no view of the vaginal walls, that it brings together (in a deceptive way) the lips of a torn cervix, that it makes it very difficult to use any other means of diagnosis in association with it (*e.g.* curved sound or volsella), and that it is useless for the performance of any of the minor operations of gynæcology (save that of applying caustic to the cervix).

(3) Valvular Variety of Vaginal Speculum: this variety, of which there are many types (Cusco's, Reid's, Barnes' Neugebauer's, and Gau's, which are bivalve, and Nott's, which is a trivalve), has much the same sphere of usefulness as the tubular.

(*a*) *Description*: Cusco's instrument consists of two metal blades, jointed to each other at their bases, and having a thumbpiece by which they can be separated, and an apparatus (travelling nut on a screw) by which the degree of separation can be regulated and fixed; in Reid's speculum the blades are separable and move on parallel bars; Neugebauer's speculum really consists of the blades of a Sims' speculum separated and acting as two.

(*b*) *Mode of Use*: (1) patient in same position as for the tubular variety; (2) speculum is warmed and has vaseline applied on the outer aspect; it is introduced closed, with its blades right and left (to suit the vaginal orifice and labia): it is held in the right hand, the fingers of the left separating the labia; when passed within the orifice, it is turned round, so that the blades are anterior and posterior (to suit vaginal walls), that with the screw being posterior; the blades are then opened and fixed by the screw; in removing it the blades must first be closed; both in introducing and in withdrawing it care must be taken not to catch vulvar hairs in it or pinch up the vaginal walls with the blades. (*Note.*—The above description applies specially to Cusco's instrument; if Neugebauer's be used, the larger blade is introduced like a Sims', the smaller is passed into the vagina along the anterior wall, and fits into the larger blade.)

(*c*) *Advantages and Disadvantages*: its advantages are those of the tubular type; so are its disadvantages; but it also causes great separation of the lips of a torn cervix, and may thus mask the lesion, and the vaginal walls may prolapse between the blades and obscure the view.

(*Note.*—The speculum, as a means of diagnosis, is more useful to the beginner than to the practised gynæcologist, who has developed his skill in the use of the vaginal and bimanual methods; educated touch will give nearly all the information obtainable by sight.)

(II.) *Volsella.*

(*a*) Description: is a pair of metal toothed forceps; the blades are separable and are secured by a pin, the handles are provided with a simple fixing apparatus (*e.g.* a metal clasp), the whole instrument has a slight pelvic curve, and each blade has one, two, or (best) three teeth; it should be from eight to nine inches in length.

(*b*) Mode of Use: (1) if it be intended to introduce the volsella by touch alone, then the patient lies on the left side, or semi-prone; the examiner passes two fingers (index and middle) of the right hand into the vagina and touches the cervix (anterior lip) with them; then, holding the volsella in his left hand, he passes it in, guiding it along the fingers of his right hand, and seizes the cervical lip with it; traction is now made (force of from three to seven pounds may be safely used), and the cervix drawn down to the vulva; (2) if it be the gynæcologist's purpose to introduce the volsella through a speculum, then the patient lies in the semi-prone or (especially for operative purposes) in the lithotomy position; the cervix is exposed and grasped by the volsella, the examiner guiding the instrument by sight. If the gynæcologist be single-handed, he may fix the volsella by a tape to the patient's dress, or to the bedclothes; with the help of the volsella, and with the patient in the semi-prone position, as good a view (or better) may be obtained of the vagina and cervix as with any speculum. For operative purposes two volsellæ may be used, one for each lip.

(*c*) Rationale of Use: the mobility of the uterus and of the anterior segment of the pelvic floor permits the downward displacement of the organ, and the small degree of sensitiveness of the cervical tissues lessens the discomfort to the patient arising from the bite of the instrument.

(*d*) Diagnostic Uses: the volsella serves—(1) for bringing parts within the range of vision, thus the cervix, vaginal roof, and vaginal walls can be directly inspected for lacerations, polypi, fistulæ, erosions, ulcerations, and the like; (2) for bringing these parts within the range of touch, aided by vision; (3) for determining the relation of abdominal tumours to the uterus, downward descent of that organ being accompanied by descent of the tumour if it be uterine, and by no change in its position

if it be not; (4) for exploring structures or organs (*e.g.* ovaries) lying behind the uterus, especially when the use of the volsella is combined with the practice of rectal touch; (5) as an aid in the passage of the uterine sound or curette.

(*e*) THERAPEUTIC USES: although we are here concerned with diagnosis, it may be borne in mind that the uses of the volsella (along with other instruments) in treatment are numerous—(1) operations on the cervix (amputation, repair of lacerations, removal of growths); (2) operations on the vaginal walls (fistulæ, curettage, colporrhaphy); (3) operations on the uterine interior (curettage, enucleation of fibroids, insertion of tents or pessaries); (4) extirpation of the uterus (hysterectomy), or of small ovarian cysts (vaginal ovariotomy); (5) exploration of the lower part of the peritoneal cavity (colpotomy); (6) replacement of retroverted uterus (*e.g.* in pregnancy); and (7) massage of the pelvic organs.

(*f*) CONTRA-INDICATIONS: (1) pregnancy (save in cases of retroversion, when may be used with care as aid in reposition of uterus); (2) menstruation; (3) acute peritonitis or cellulitis; (4) dilated Fallopian tubes, especially in pyosalpinx; (5) hæmatocele and tubal gestation; (6) cancer of the cervix.

(II.*) *TENACULUM*: little used nowadays.

(*a*) DESCRIPTION: the tenaculum consists of a metal rod tapering to the end, the end being a sharp hook.

(*b*) USES: all the purposes for which it has been recommended are better performed by the volsella, save perhaps its employment in operations for vesico-vaginal fistulæ. Two tenacula may be used as a volsella in diagnosing cervical tears.

(III.) *UTERINE SOUND.*

(*a*) HISTORICAL NOTE: a uterine sound is said to have been known to the ancients, but this is doubtful, as a clear distinction was not drawn by them between the os uteri and the orifice of the vagina. In 1843, J. Y. Simpson (Edinburgh), Huguier (Paris), and Kiwisch (Prague), almost simultaneously invented an instrument for sounding the uterine interior, and the first (Simpson) effectually established its value by abundant clinical evidence; its introduction marked a great advance in the perfection of gynæcological diagnosis.

(*b*) DESCRIPTION: (1) J. Y. Simpson's sound was a curved rod of German silver, 12 inches long, with a rounded end, and with an oval handle roughened on one side (that which corresponded with the concavity of the curve of the stem); the rod had several markings on it at $1\frac{1}{2}$, $2\frac{1}{2}$, $3\frac{1}{2}$, $4\frac{1}{2}$, $5\frac{1}{2}$, $6\frac{1}{2}$, $7\frac{1}{2}$, and $8\frac{1}{2}$ inches from the point; (2) A. R. Simpson's sound is more useful; it is a rod of copper, nickel-plated, about 3 inches shorter, with a smaller, and nearly square, handle, and with only four markings on it (a circular ridge at $2\frac{1}{2}$, a groove at $3\frac{1}{2}$, a double ridge at $4\frac{1}{2}$, and a groove at $5\frac{1}{2}$ inches respectively); these modifications enable the gynæcologist to use the sound in association with the bimanual examination, increase its pliability, and diminish the risk of fracture near the point; the uterine probe (*e.g.* Playfair's) is simply a modified sound (lighter and more pliable).

(*c*) CONTRA-INDICATIONS: (1) *Pregnancy*; (2) Menstruation; (3) Acute inflammation of uterus, ovaries, or pelvic peritoneum; and (4) Cancer of cervix or body of uterus.

(*d*) DANGERS: (1) *Production of Abortion*; (2) introduction of sepsis; (3) perforation of uterine wall.

(*e*) PRELIMINARY PRECAUTIONS: the dangers being what they are, it is necessary—(1) to ascertain carefully the date of the last menstruation, and if the patient has passed one or more periods, to refrain from examining with the sound, lest she be pregnant, and so the gynæcologist unwittingly cause abortion; (2) to perform a careful bimanual for the same purpose (to exclude pregnancy), and to get an idea of the direction of the uterus. (*Note.*—The patient may mislead the gynæcologist either intentionally or unintentionally in regard to her menstrual dates); (3) to sterilise the instrument, and to cleanse the patient's vulva and vagina; (4) give the sound the curve which the bimanual has shown to be necessary.

(*f*) MODE OF USE: (1) *by touch alone*: with the patient in the dorsal, left lateral, or semiprone position, the gynæcologist introduces the forefinger of his right hand into the vagina and touches the anterior lip of the cervix; then holding the sound in his left hand, he passes it up along the vaginal finger until the point enters the os: (α) if he has ascertained (by the bimanual) that the uterus is retroverted, he now simply carries the handle of the sound forwards, when, almost by its own weight, the instrument passes into the interior of uterus; (β) if, however, the uterus lie to the front, then the sound requires to be rotated, so as to make the concavity of the curve look forwards, and this is done by carrying the handle round "the arc of a wide semicircle" (till the rough surface on it is anterior), and then bringing the handle back to the perineum, when the instrument ought (without any force) to slip into the uterine cavity. (*Note.*—In this second case the forefinger of the left hand may be passed into the vagina till it touch the posterior lip of the cervix, and the sound (held in the right hand) be passed inwards and onwards without the necessity of the handle revolution.)

(2) *By sight and touch*: when the introduction of the sound is difficult (*e.g.* acute flexion of uterus), or when the gynæcologist is a beginner, it is well to pass the Sims' speculum,

draw down (and so straighten) the uterus with a volsella, and then, holding the sound in the right hand, simply to allow it to pass into the uterus by its own weight, guiding it with sight and touch combined. (*Note.*—If the cervical canal be tortuous, or be blocked with a growth (*e.g.* a fibroid tumour), several attempts may be necessary before the introduction can be accomplished.)

(*g*) Diagnostic Uses:

(1) Condition of os uteri and cervical canal (*e.g.* atresia, narrowness at external or internal os).

(2) Length of uterine cavity (*e.g.* increased (more than $2\frac{1}{2}$ inches) in subinvolution, fibroids, hypertrophy of cervix, endometritis, prolapsus; lessened (less than $2\frac{1}{2}$ inches) in superinvolution, infantile uterus).

(3) Direction of uterine axis (*e.g.* retroversion, anteversion, lateriversion, especially before curettage).

(4) Relation of axis of cervix to axis of body (*e.g.* retroflexion and anteflexion).

(5) Mobility of uterus.

(6) Relation of uterus to abdominal tumours (*e.g.* if tumour be uterine, its palpation through abdominal wall conveys impulse to the sound).

(7) Condition of endometrium (*e.g.* if there is endometritis the presence of sound will cause pain, and the instrument may be blood-stained when withdrawn).

(8) Differential diagnosis between polypus in uterus (when the sound passes to normal distance or farther) and inversion of uterus (when it is arrested inside cervix).

(9) To aid the student in learning the bimanual (under teacher's supervision).

(*Note.*—When, after meeting with resistance, the sound suddenly passes in for a distance of some inches, either—(1) perforation of the uterine wall has occurred; or (2) the instrument has passed along a Fallopian tube; or (3) a sudden state of uterine inertia with enlargement of the cavity has developed.)

(*h*) Therapeutic Uses:

(1) Reposition of displaced movable uterus (especially in retroversion); comparatively rare now, as bimanual reposition is more practised.

(2) Rectification of a flexion (requires care, and sound has to be specially (sharply) curved).

(3) Dilatation of narrow os or cervical canal, especially in cases of poorly developed uterus, with scanty and painful menstruation.

(4) Making applications (caustic) to endometrium, the sound being dressed with cotton wool (Playfair's probe serves better) after curettage.

(*i*) Estimate of Present Value of Sound: since the bimanual method of examination has been perfected and commonly practised, the use of the sound has become more restricted; but it is still of value when it is difficult to define the outlines of the uterus, and especially of its fundus, by the bimanual alone; in combination with the bimanual it is of great assistance in enabling the gynæcologist to educate himself to the significance of the sensation conveyed to his fingers.

(IV.) *Uterine Curette* (*see also* Curettage).

(*a*) Historical Note: introduced by Récamier, in 1850, for the purpose of scraping away fungosities from the interior of the uterus; Sims used it for same purpose (1865) after previous dilatation with a sponge tent; in 1874 Thomas invented his dull wire curette, and Mundé in 1878 popularised its use and emphasised its value in diagnosis rather than in treatment; since 1878 its sphere of usefulness has been greatly widened, and it is now more used in treatment than in diagnosis.

(*b*) Description: many varieties of curette, but it is essentially a metal rod with a loop of wire, or a nail- or spoon-shaped scraping surface at its distal end; it sometimes has a knob on the stem $2\frac{1}{2}$ inches from the end, to mark the normal length of the uterus; and occasionally it has the handle and stem hollow, so as to allow a stream of fluid to run through it during curettage ("flushing" curette); Martin's curette, or that of Récamier-Roux, is a useful form.

(*c*) Mode of Use: with the patient in the semi-prone or lithotomy position, and under an anæsthetic, the operator passes the Sims' speculum, fixes the cervix with a volsella, and then dilates the cervical canal with Auvard's, Hegar's, or Ellinger's dilators, until it is wide enough to allow the curette to pass easily in; he next introduces the curette and scrapes the uterine wall with it (thoroughly if for treatment, locally if for diagnosis); the uterine interior can then be washed out, and have a caustic application (*e.g.* iodised phenol) made to it (on a dressed sound or Playfair probe). Further details are given in the article, Curettage, Uterine.

(*d*) Indications:

(1) For *diagnosis*: a scraping from the interior of the uterus being obtained for microscopic examination, as in cases where the diagnosis between endometritis and carcinoma, sarcoma, and chorion-epithelioma of the uterus is in doubt.

(2) For *treatment*: in a large number of morbid states, but chiefly in (α) retained products of conception, setting up hæmorrhage or sepsis, (β) endometritis, especially the hypertrophic form, (γ) some infective states of the uterus (septic and gonorrhœal), (δ) uterine cancer (as a palliative in non-operable cases), (ϵ) hysterectomy for uterine tumours (as a preparatory stage in the operation), and (ζ) rarely in uterine fibroids (for the hæmorrhage).

(*e*) Dangers: these are (1) production of abortion, (2) introduction of sepsis, (3) perforation of uterus, (4) subsequent obliteration of

the uterine cavity, or superinvolution (rare) from adhesive inflammation, and (5) hæmorrhage (rare).

(*f*) Precautions : (1) eliminate possibility of pregnancy, (2) establish strict asepsis in and after operation, (3) avoidance of force, and (4) rest in bed for patient for a day or two before operation, for a week after it, and at the next menstrual period.

(V.) *Aspiratory Needle.*

Occasionally, when a fluid collection in the abdomen or pelvis (especially in one of the broad ligaments) has been diagnosed, it may be well to discover its nature (before operation) by aspirating some of it through the vaginal roof or even through the abdominal wall. An ordinary hypodermic needle may be used, or Bartlett's exploring aspirator, or a Pravaz syringe. In this way fluid for chemical, bacteriological, and microscopical examination can be obtained ; and Fehling (1902) claims for the method important diagnostic results.

(VI.) *Cervical Dilators.*

(1) *Rapid Dilatation* with metal or hard rubber dilators.

(*a*) Description : (i.) the gynæcologist may use a number (24) of metal rods about six inches long, and having a diameter increasing in a graduated manner from $\frac{1}{12}$ inch to about $1\frac{1}{12}$ inch ; such have a short flat handle and are slightly curved ; (ii.) similar rods in hard rubber are the well-known Hegar's dilators ; (iii.) metal dilators with expanding blades (two, or better, three in number) have also been used, those of Ellinger, Goodell, Sims, and Schultze being the best known ; Bossi's large metal dilator is not so useful in gynæcological as it is in obstetric practice.

(*b*) Mode of Use : (1) if the graduated rods be employed the patient is anæsthetised, the vagina rendered aseptic, and the cervix fixed with two volsellæ ; the sound is passed to determine the direction and length of the uterine cavity ; then the dilators, sterilised and coated with vaseline, are passed in (just as the sound is) one after another, beginning with one a little larger than the ordinary uterine sound ; dilatation, sufficient to allow the passage of the index finger, ought to be attained in from fifteen to twenty minutes ; (ii.) if the expanding metal instrument be employed the preliminaries are the same, and then the dilator is passed into the cervix with the blades closed ; the handles are then slowly approximated, causing gradually separation of the blade and dilatation of the canal.

(2) *Slow Dilatation with Tents :*

(*a*) Historical Note : tents, first made of sponge, were introduced by J. Y. Simpson, who had noted the dilating effect upon the cervical canal of a polypus growing down into it, and was led to devise a means of similarly dilating it by an enlarging body inserted into it from below.

(*b*) Description : Tents are conical bodies, a little longer than the cervical canal, and differing slightly in calibre ; the material may be sponge (dried, compressed, impregnated with an antiseptic, covered with grease, and having a tape at the proximal end) or sea tangle (*laminaria digitata*), or tupelo (*nyssa aquatilis*) ; of these tupelo is generally preferred, for it absorbs fluid more readily, and so expands more quickly and to a greater degree than the other two.

(*c*) Mode of Use : (i.) Preliminary Precautions : patient in bed in her home or in hospital (do not introduce tents in consulting-room, as was formerly done) ; vagina is douched with antiseptic lotion before and after introduction of tent ; prepare tents (only tupelo and tangle are now used) by dipping them in strong antiseptic lotion, and then in sterilised water ; patient's vulva is to be sterilised, as also operator's hands ; (ii.) place patient in semi-prone or lithotomy posture, pass Sims' speculum, draw down cervix by means of volsella attached to anterior lip, and pass the tent, on a sound or tent-carrier or held in a pair of forceps, into the cervical canal (to introduce a tent with the fingers alone, and without seeing the cervix, is not to be recommended) ; an iodoform gauze plug is placed in the vagina ; (iii.) the tent may be left in for twelve hours, and a qualified attendant must be within easy reach of the patient during this time, and a vaginal douche every four hours may be given and the plug replaced ; it is to be withdrawn by gentle traction on the string attached to its base ; (iv.) a second tent may be used after the first, but with care.

(*d*) Indications : (1) tents are seldom used nowadays for diagnosis alone ; (2) almost their sole use is to restrain hæmorrhage and dilate the cervix to permit of active treatment in cases of abortion, and more especially in molar pregnancies (*e.g.* hydatid mole).

(*e*) Dangers and Contra-indications : (1) the chief danger is sepsis, and it is a very real one, especially with sponge tents ; (2) the contra-indications are pelvic inflammation (including oophoritis and salpingitis), cancer of the cervix, and hæmatocele.

8. Physical Examination of the other Systems of the Body (Nervous, Respiratory, Circulatory, Digestive, Urinary, Cutaneous, and Locomotory).

(i.) Although the gynæcologist's attention is specially focussed upon the physical examination of the genital organs, he must not neglect altogether that of the *other systems of the body.* For instance the examination of the blood may reveal a leucocytosis, indicating deep-seated suppuration in the pelvis, and may even help in the differential diagnosis of malignant from simple ovarian cysts ; the examination of the

urine (for albumin, sugar, etc.) is often of great value; the bacteriological investigation of discharges from the uterus and vagina may be helpful; and the various organs of the different systems ought to be examined in order to separate morbid states which are genital reflexes from those due to actual disease of these organs.

(ii.) Examination of the Urinary Tract: although not strictly within the province of the gynæcologist, the bladder and ureters are nowadays often examined by him.

(1) *Palpation of Urethra and Bladder and Ureters*: (*a*) during the vaginal examination the whole of the urethra can be palpated by the finger pressing on the anterior vaginal wall (the consistence, thickness, tenderness, and mobility of it can be thus made out), and by pressure any secretion may be squeezed out of the meatus and examined microscopically and bacteriologically; (*b*) the base of the bladder can also be palpated (the finger being in the anterior fornix vaginæ); and by the bimanual some notion may be gained regarding the state of the vesical walls and the presence of foreign bodies (*e.g.* calculi) in the viscus; (*c*) the ureters, if thickened and hardened, may be felt per vaginam in their lower pelvic portion and per rectum in their upper pelvic part. (*Note.*—Digital palpation of the bladder per urethram (after digital dilatation) is now hardly ever practised; but some information regarding the interior of the organ and its contents may be got by the use of the sound.)

(2) *Inspection* (*Cystoscopic*) *of the Bladder and Urethra*: (*a*) Preliminaries are the emptying of the rectum and bladder and thorough cleansing of the external parts, and especially of the vestibule and meatus urinarius; sterilisation of the instruments (dilators, cystoscopes, and obturators, ureteral catheters, etc.), and of examiner's hands; (*b*) position of patient is either genu-pectoral, or (better) exaggerated lithotomy, the hips being elevated on bran bags six inches above the level of the table; (*c*) anæsthetic may be chloroform or the local application (to interior of urethra) of cotton wool soaked in cocain solution (10 per cent); (*d*) urethra is dilated with Hegar's or a conical dilator up to a diameter of from 12 to 15 mm.; (*e*) Kelly's cystocope (nickel-plated cylinder, 8 cm. long, and having diameter of from 10 to 12 mm.), with obturator in it and well lubricated, is pushed into bladder; (*f*) when the obturator is withdrawn, air will rush into and distend the bladder; (*g*) light (from an electric lamp and mirror) can then be thrown into the bladder, and the interior inspected (noting the general appearance of the mucous membrane, the two ureteral eminences and folds, the interureteric fold, and the internal urethral orifice); the internal urethral orifice and the urethra itself can be inspected by slowly withdrawing the cystoscope; (*h*) it may be necessary to remove some drops of urine by a suction apparatus (tube and suction bulb).

(3) *Catheterisation of the Ureters*: (*a*) during the cystoscopic examination of the bladder the ureters may be sounded; (*b*) the method is as follows:—one ureteric opening is located (by slightly inclining the cystoscope to one side), any secretion is removed from it by cotton held in a pair of delicate mouse-tooth forceps, then a long or short ureteral catheter (sterilised) is passed through the cystoscope and guided along the ureter up into the pelvis of the kidney (the stylet being withdrawn by an assistant as the catheter advances); (*c*) the process is repeated for the opposite ureter, but with a different catheter (to avoid infection of one ureter from the other); (*d*) in this way ureteric strictures, twists, and calculi can be discovered, the urine got for examination from each kidney, and the ureters can be exactly defined prior to such operations as hysterectomy (*Note.*—By means of the Harris urine segregator (a double catheter with a forked end) the urine from each kidney can be collected separately without the ureters being entered, but not with such exactness as it is by ureteral catheterisation); (*e*) avoid catheterisation of the ureters when there is septic cystitis or tuberculosis of the bladder.

9. Diagnostic Operations: in addition to curettage and cervical dilatation, which are really operative procedures, even when the object is purely diagnostic, it may be noted that abdominal and vaginal sections are sometimes performed for the purpose of ascertaining the nature of a morbid state ("exploratory section").

Note.—Illustrations of most of the instruments described in this article will be found in the plate accompanying Curettage (vol. ii. p. 260).

Gynandry. — Pseudo-hermaphroditismus femininus; an individual with ovaries who yet has certain male characters is called a gynander; gynandry is the antonym of androgyny. *See* Hermaphroditism; Uterus, Malformations of (*Gynandria*).

Gyniatrics.—The therapeutics of gynæcology, or simply gynæcology itself as distinguished from obstetrics.

Gynocardia Oil.—Chaulmoogra oil or oleum gynocardiæ. *See* Chaulmoogra Oil; Leprosy (*Treatment*).

Gyropsoriasis.—Psoriasis in which the patches have a gyrate appearance.

Gyrospasm. *See* Head-Shaking (*Synonyms*).

Gyrus.—A convolution (*e.g.* of the brain) or a winding (as of the cochlea). See Brain, Physiology of (*Gyrus Fornicatus*); Physiology, Nervous System (*Brain*).

Haab's Macular Disease. *See* Eyeball, Injuries of (*Injuries by contusion, Rupture of the Choroid*).

Haarlem. *See* Balneology (*Holland*).

Habit. *See* Insanity, Nature and Symptoms (*Index of Mental Functions, Habit*); Nose, Post-nasal Adenoid Growths (*Habitual Mouth Breathing*).

Habit Chorea or **Habit Spasm.**—A form of chorea, also known as convulsive tic. *See* Chorea (Diagnosis); Rheumatism in Children (*Symptoms, Nervous*).

Habits. *See* Morphinomania and allied Drug Habits.

Habitual Abortion. *See* Abortion (*Recurrent*).

Habitual Drunkards. *See* Alcoholism (*Treatment*).

Habitus.—The external indication of a constitutional morbid state, such as the apoplectic habitus, or the habitus phthisicus.

Hacker's (Von) Operation.—Posterior gastro-enterostomy. *See* Stomach, Surgical Affections of (*Operations on the Stomach*).

Hacking Cough.—A shallow, frequent, and (usually) dry cough.

Hacking Movements.—Strokes or beats, in use in massage, applied either by the finger tips, or the ulnar side of the hand, or the dorsal surface of the three middle fingers.

Haddock. *See* Invalid Feeding (*Food for the Aged, Fish*).

Hæma.—In compound words *hæma-* signifies relating to the blood (Gr. αἷμα, blood), but it is an incorrect form, and *hæmato-* or *hæmo-* ought to be employed; *e.g. hæmochroses*, or diseases in which the colour of the blood is changed.

Hæmacytometer.—An instrument for ascertaining the number of red corpuscles in the blood, more correctly *hæmatocytometer*.

Hæmadipsia Ceylonica.—A small land leech which draws blood through the skin, it may be with fatal effects.

Hæmagglutinins. *See* Agglutination; Immunity; Typhoid Fever (*Pathology*).

Hæmagogue.—A medicine promoting the menstrual discharge, or a hæmorrhoidal flux.

Hæmalbumin.—A preparation said to contain the salts and albuminoids of the blood.

Hæmalopia or **Hæmalops.**—A condition of the eye in which everything appears to have a blood-red colour; or, simply, an effusion of blood into the eye.

Hæmamœba and **Hæmamœbiasis.** *See* Malaria (*Synonyms*); Parasite (*Protozoa, Hæmosporidia, Hæmamœba Danilewskii*).

Hæmangioma.—A tumour or formation made up almost entirely of blood-vessels. *See* Tumours (*Angeiomata*).

Hæmaphæin.—A brown colouring matter of the blood, probably due to decomposition of hæmatin.

Hæmarthrosis.—An effusion of blood into the cavity of a joint. *See* Hæmophilia (*Joint Affections*); Joints, Diseases of (*Hæmarthrosis, Bleeders' Joint*); Knee-Joint, Injuries of (*Wounds*).

Hæmatamœba. *See* Hæmamœba.

Hæmatelytrometra.—An accumulation of menstrual blood in the vagina and uterus, usually on account of the presence of an imperforate hymen or vaginal atresia.

Hæmatemesis.

See also Aorta, Aneurysm and Dilation (*Course and Terminations, Rupture*); Gastro-Intestinal Disorders of Infancy (*Hæmatemesis*); Gelatin; Hæmoptysis (*Diagnosis*); Hysteria (*Disorders of the Digestive System*); Liver, Diseases of (*Hypertrophic Biliary Cirrhosis, Physical Signs*); Liver, Diseases of (*Signs and Symptoms of Cirrhosis, Hæmatemesis*); Liver, Diseases of (*Portal Thrombosis, Symptoms*); Malingering (*Digestive System and Alimentary Canal*); Melæna (*Surgical Treatment*); Myiasis (*Intestinal Symptoms*); New-born infant (*Diseases, Melæna*); Spleen, Surgery of (*Obliteration of Splenic Vein, Hæmatemesis*); Stomach and Duodenum, Diseases of (*Special Symptomatology of Ulcer of the Stomach, Vomiting*).

Hæmatemesis, or vomiting of blood from the stomach, must, in the first instance, be distinguished from the discharge by the mouth of blood derived from other sources. The commonest and most important of these is hæmo-

ptysis; here the blood is of a bright scarlet colour, alkaline in reaction, frothy from admixture with air, and comes up with a cough, and for days afterwards the sputum, if there be any, is tinged with blood. It is, however, possible in profuse hæmoptysis for some of the blood to be swallowed and so give rise to secondary hæmatemesis. In epistaxis, or in fracture of the base of the skull, if the patient be unconscious, the blood may run down into the œsophagus and be swallowed, so that it enters the stomach, and when rejected is hæmatemesis. When the patient is conscious the blood may pass forward into the mouth and be spat out. Oozing from the gums, carious teeth, tonsils, or pharynx in various diseases may again sometimes imitate slight hæmatemesis. Anæmic girls not very infrequently speak of having brought up blood, which on further investigation proves to be very small in amount, and to occur in the early morning. In these cases the blood is probably provided by the nasopharynx.

Feigned or hysterical hæmatemesis is sometimes due to industrious gum-sucking, to self-made wounds, or to extraneous blood or coloured fluids introduced into the mouth. The patient's manner and behaviour may arouse suspicion. The mouth and hands should be carefully examined for bleeding points, and the vomit submitted to microscopical and chemical tests for blood.

When blood is poured out into the œsophagus it may run down into the stomach, and when subsequently vomited is indistinguishable from ordinary hæmatemesis; this is particularly likely to occur when the source of the hemorrhage is an ulcerated varicose vein near the lower end of the œsophagus. Sometimes when bleeding takes place from the walls of the œsophagus the blood does not enter the stomach, but wells up without any retching or vomiting. This is sometimes seen in ulcerated varicose veins, or in aortic aneurysm rupturing into the œsophagus; in both of these conditions the hæmorrhage may be very profuse. Small quantities of blood mixed with froth and mucus may come from the œsophagus in acute traumatic œsophagitis due to the ingestion of corrosive or irritant poisons, and is of course accompanied by great dysphagia. In rare instances blood and pus mixed together are hawked up from the œsophagus, and are due to one of the rare conditions, phlegmonous œsophagitis, or an abscess opening into the gullet. When simple ulceration of the mucous membrane of the œsophagus is present, vomiting from other causes may be streaked with blood.

Hæmorrhage is occasionally seen in the course of malignant disease of the œsophagus, which ulcerates early and produces symptoms of œsophageal obstruction. But it is noteworthy how extremely seldom carcinoma of the œsophagus ulcerates into the aorta, although their anatomical relationship is very intimate.

True Hæmatemesis.—*Characters*.—The blood which is forcibly vomited up may be expelled through the nose as well as the mouth. Shortly before its onset the patient may feel distended, uncomfortable, faint, and become blanched.

As the vomited blood passes over the orifice of the larynx cough may be set up. This must be borne in mind, as otherwise this association of cough and blood may be regarded as proof of hæmoptysis. The cough follows the hæmatemesis, whereas in cases of copious hæmoptysis when some of the blood is swallowed the cough precedes the hæmatemesis.

Colour.—The blood is usually more or less altered in colour, being darker than the action of the gastric juice; on standing, however, it tends to pick up oxygen from the air, and so to become of a brighter hue; it is, accordingly, important that its naked-eye appearances should be noticed at once. It will probably be acid in reaction, and may be mixed with food.

When in copious hæmatemesis the blood is of an arterial character, rapid hæmorrhage from an artery exposed in a gastric ulcer has probably taken place. When, on the other hand, it is black, clotted, and copious, the bleeding probably depends on cirrhosis of the liver. Small "coffee ground" vomit is especially associated with carcinoma of the stomach; the blood is then very extensively acted upon by the gastric juice, and some question may arise as to whether the coffee ground vomit is blood or some article of food, drink, or medicine, such as coffee, porter, beef-tea, charcoal, or even the mixture of tea and perchloride of iron (Bramwell). In such a case the hæmin test with salt and hydrochloric acid, or the spectroscopic test, should be employed, as being more reliable than microscopic examination, inasmuch as the corpuscles may be digested and destroyed.

Frequency.—Repeated large hæmorrhages may be due to ulcerated varicose veins at the lower end of the œsophagus in cirrhosis, to minute pore-like erosions of the gastric mucous membrane, or to gastric ulcer, and may so exhaust the patient as to be fatal. A single hæmatemesis is rarely fatal except when a large aneurysm bursts into the stomach or œsophagus—a rather rare event. It has, however, occurred in some cases of ulcerated œsophageal piles.

Small coffee-ground vomiting, especially when repeated, points to carcinoma of the stomach; here vomiting is reflex, and depends on the irritation of the growth. Any blood that happens to be on the stomach is accordingly brought up with the other gastric contents; in large hæmatemesis, as in gastric ulcer, the vomiting is the result of stimulation of the gastric muscular walls by the distension of the organ, and is comparable to the action of an enema on the lower bowel.

In very rare instances hæmorrhages may recur at intervals over a period of years with good health in between; this has been observed in splenic anæmia (Osler), and exceptionally in cirrhosis from varicose veins at the lower end of the œsophagus (Garland).

Causes and Source.—The blood may come from the œsophagus or duodenum as well as from the stomach itself.

Varicose œsophageal veins are generally part of the compensatory venous anastomosis set up in hepatic cirrhosis, but they may also develop in extensive malignant infiltration—for example, in secondary melanotic sarcoma of the liver, where this induces portal obstruction, or they may be idiopathic, viz. without any causative lesion.

As a result of chronic inflammation the varicose veins tend to become adherent to the mucous membrane, and ulceration may follow with profuse or even fatal hæmorrhage. This event may come on without any previous symptoms of ill-health, the underlying cirrhosis being quite latent.

Duodenal ulcer may give rise to regurgitation of blood into the stomach and hæmatemesis. Ordinary duodenal ulcers are found in the first part of the duodenum; hæmorrhage may also occur from the ulceration and passage of gall-stones into the duodenum. An abdominal aneurysm rupturing into the duodenum has been known to give rise to fatal hæmatemesis, while an hepatic aneurysm bursting into the bile duct may also be a cause of hæmatemesis, though melæna alone is more often recorded.

In the stomach the lesions that give rise to hæmatemesis may be divided into

(1) Gross lesions.

(2) More minute lesions of the mucous membrane.

(i.) *Gross Lesions.*—Gastric ulcer is more frequent in anæmic young women, and is then commonest near the pylorus. It also occurs in men at a more advanced age, and is often more extensive and firmly adherent to neighbouring viscera than in the first category; it may be situated in any part of the stomach. Repeated and large hæmorrhages may occur from minute abrasions of the gastric mucous membrane, "pore-like erosions," or "exulceratio simplex."

Small ulcers may supervene in chronic engorgements of the stomach as the result of localised hæmorrhages into the mucous coat. The ulceration may open up a vessel and give rise to profuse hæmatemesis, and from their minute size they may easily be overlooked. Possibly some cases of fatal hæmatemesis, where the stomach has been described as perfectly healthy, are of this category. These small ulcers are often seen near the cardiac orifice; in the passive congestion due to cirrhosis these ulcers usually open up a vein, while in that due to cardiac disease an artery is eroded (S. Fenwick).

Varicose veins of the stomach are much rarer than in the lower part of the œsophagus, but may give rise to severe hæmatemesis.

Other rare causes of hæmatemesis are aneurysms of the gastric, splenic, or hepatic arteries opening into the stomach. Carcinoma of the stomach may arise at the cardiac orifice, at the cardiac end, at the pylorus, or may involve the whole of the organ. The hæmorrhage is usually slight, rarely profuse, and, as already pointed out, the blood is apt to be retained in the stomach, and as a result of the action of the gastric juice become black or "coffee ground" in appearance. In very rare instances a malignant growth may invade the stomach from without, and give rise to hæmatemesis. The writer has seen this in a case of carcinoma of the left suprarenal body.

(ii.) More minute lesions of the gastric mucous membrane occur in acute gastritis. This may be of local origin, and follow the ingestion of toxic fluids, such as large quantities of spirit, acids, phosphorus, or other poisons.

In chronic enlargement of the stomach depending on the backward pressure of heart disease, hepatic cirrhosis, thrombosis of the portal vein, or compression of the gastric veins by adhesions, inflammatory and degenerative changes in the mucous membrane of the stomach are readily set up, with the result that oozing of blood follows. In chronic engorgement of the stomach the further change of gastritis may be readily set up by swallowing the pus and micro-organisms present in dental caries and pyorrhœa alveolaris. In cirrhosis of the liver it is generally assumed that hæmatemesis may be due to a widespread venous or capillary oozing, but it is probable that in addition to the venous stasis some degenerative changes in the mucous membrane are necessary to allow of the extravasation of blood. In splenic anæmia, however, hæmatemesis has been explained by Osler as being purely mechanical and due to venous engorgement.

Changes in the gastric mucous membrane may be part of a general condition, and may be secondary to severe hæmic infections or intoxications, such as yellow fever, icterus gravis, acute yellow atrophy of the liver, and the hæmorrhagic or malignant forms of the specific fevers. In these, and sometimes in pyæmic and septicæmic states, blood may be extravasated in varying amounts into the stomach. In diphtheria marked degeneration of the gastric mucous membrane is met with, and blood may be found in the vomit in cases that are not of the hæmorrhagic type.

Hæmatemesis is very rare in typhoid fever, but has been known to occur and is correlated with the rare lesions of typhoid ulcers in that organ. In a few instances pneumonia is accompanied by hæmatemesis, due to secondary ulceration in the stomach or duodenum. Hæma-

temesis may occur in hæmorrhagic states, such as purpura hæmorrhagica, leukæmia, pernicious anæmia, and hæmophilia. It has also been observed in association with urticaria, and the two are doubtless manifestations of the same underlying blood state.

Hæmatemesis is said to represent menstruation. It is, however, quite possible that the explanation of some of these cases of vicarious menstruation is that there is a chronic gastric ulcer which periodically bleeds, while in others the hæmorrhage may have been feigned. Vicarious menstruation was recognised by Sir Thomas Watson, but at the present time most practising physicians will endorse the late Dr. J. Matthews Duncan's dictum—"I have all my life been on the look-out for it" (vicarious menstruation), "but I have never seen an example, and do not expect to do so."

Diagnosis.—Sudden hæmatemesis without any history of previous illness is in a middle-aged patient most likely to be due to latent cirrhosis of the liver. If the liver and spleen are found to be enlarged, and the individual's habits are known to be alcoholic, the diagnosis is strengthened. If the sudden hæmorrhage is accompanied by the rapid development of ascites, thrombosis of the portal vein should be thought of. Great enlargement of the spleen would point to spleno-medullary leukæmia or splenic anæmia, and a blood examination should be made to elucidate the diagnosis.

A large hæmatemesis may occur from acute gastritis due to recent alcoholic excess, and the question will arise as to whether there is latent cirrhosis as well. The absence of any evidence of cirrhosis and the occurrence of hæmatemesis in a young man are in favour of alcoholic gastritis.

If there has been dyspepsia for some time before the onset of hæmatemesis the cause may be gastric ulcer, carcinoma of the stomach, cirrhosis, or duodenal ulcer. If the patient is a young anæmic woman, and the hæmorrhage is copious, the probabilities are strongly in favour of a gastric ulcer; this will be supported by a history of pain, which coming on directly after food is relieved by vomiting, and by finding a point of maximum tenderness on deep pressure over the stomach; with simple pore-like erosions of the gastric mucous membrane, tenderness, however, may be absent, though the hæmatemesis may be frequently repeated and severe. Examination of the vomited matters, apart from hæmatemesis, will show an excess of hydrochloric acid in gastric ulcer. Melæna follows hæmatemesis. In very rare cases of hour-glass stomach with an ulcer in the distal pouch, melæna may occur without hæmatemesis. In other cases where the hæmorrhage is slight, melæna alone may be noted.

Gastric ulcer in men is met with later in life than in the female sex; it is accompanied by more pain, gives rise to anæmia, and may be associated with arterio-sclerosis. Care must be taken to eliminate carcinoma, cirrhosis, and duodenal ulcer.

It should be remembered that carcinoma may develop in a site of a chronic gastric ulcer, and that as this change takes place a corresponding modification in the symptoms may be observed.

In carcinoma of the stomach the vomited blood is black, resembles "coffee grounds," and is usually small in quantity, so that melæna is not noticed. If tested, the vomit will probably be found not to contain hydrochloric acid. A most important point in the diagnosis of gastric carcinoma is the presence of a tumour in the stomach or in its neighbourhood. Thus a tumour may be felt near or at the umbilicus, in the line of the falciform ligament, or on the surface of the liver. The age of the patient has a very definite bearing; ulcer is common in early life, while carcinoma is rare.

Hæmatemesis in cirrhosis may be preceded by chronic dyspepsia, but the symptoms are not so severe as in ulcer or carcinoma, and tenderness, if present, is general, and much less than the localised pain elicited on pressing over a gastric ulcer.

In duodenal ulcer the pain and dyspepsia should come on two hours after food, *i.e.* when the pylorus allows the contents of the stomach to pass into the duodenum, tenderness is more to the right of the middle line than in gastric ulcer, and the patient is nearly always a man. Melæna may precede hæmatemesis, or even occur without hæmatemesis.

When hæmatemesis is associated with jaundice, acute yellow atrophy of the liver, phosphorus poisoning, or cholæmia from the continued effects of obstructive jaundice should be thought of.

Hæmatemesis with fever suggests some severe infection, while hæmorrhage elsewhere points to a blood condition such as purpura, the malignant or hæmorrhagic forms of the specific fevers, pernicious anæmia, etc.

Prognosis.—Hæmatemesis is rarely immediately fatal; when it is so in cirrhosis, the first hæmorrhage is in a considerable proportion followed by death (Preble). The rupture of an abdominal aneurysm is, of course, likely to be fatal at once, while the copious hæmorrhage from an ulcerated œsophageal varix, or from a large artery like the splenic opened by a gastric ulcer, is much more prone to be followed by immediate grave results than the small hæmorrhages of malignant disease. When large hæmorrhages occur at short intervals death may occur from exhaustion; in such cases the stomach should be opened and the bleeding point surgically treated.

Hæmatemesis in yellow fever and in the malignant forms of the specific fevers is of

course indicative of the worst prognosis, as showing the virulence of the primary disease, while blood in the vomit of cases of diphtheria without hæmorrhages elsewhere is a very ominous sign, inasmuch as death from cardiac failure is very likely to follow, the heart muscle, like the stomach, having undergone toxic degeneration.

The more remote prognosis is that of the morbid lesion responsible for the hæmatemesis; thus the small "coffee-ground" vomit of malignant disease is of infinitely more serious import than the copious hæmatemesis of gastric ulcer.

Treatment.—The patient should be kept absolutely at rest, and the head flat so as to prevent syncope. Mental anxiety may be relieved by the hypodermic injection of morphia.

Nothing should be given by the mouth, and thirst, which after a large hæmorrhage may be urgent, should be relieved by enemata of 8 to 10 oz. of water every four hours.

Various styptics may be given by the mouth for their local action on the stomach, such as acetate of lead 2 grs. every three hours; gallic acid grs. x., combined with dilute sulphuric acid ♏x.; opium in various forms, oil of turpentine ♏xx. every six hours. I have found half-drachm doses of Ruspini's styptic in an ounce of water act well; suprarenal extract by the mouth has recently been suggested as a local styptic. In order to increase the coagulating power of the blood, calcium chloride in full doses (grs. xxx.) every two hours for a few doses may be tried. The hypodermic injection of gelatine has been employed with the same object.

The hypodermic injection of ergotin or digitalin is not advisable. The local application of an ice-bag over the stomach has been widely recommended, does not appear to do any harm, and may do good in several ways.

In cases with very severe anæmia and collapse, intravenous transfusion of saline solution should be resorted to. When large hæmorrhages are repeated at short intervals, or small hæmorrhages occur very frequently, the abdomen should be opened, the stomach incised, and the mucous membrane examined; any ulcer or pore-like erosion should be treated surgically.

In repeated hæmatemesis of splenic anæmia splenectomy has been known to prove a curative measure.

It is advisable to give a purge, blue pill, and haustus sennæ two days after hæmatemesis due to cirrhosis; in cases of ulcer it is wisest to be content with enemata.

The dietetic and further treatment varies with the cause; thus in gastric ulcer, rectal feeding should be the rule for about two weeks, whereas in cirrhosis liquid food may be cautiously given, provided there is no recurrence two or three days after the hæmatemesis. In the smaller hæmatemesis of malignant disease and that symptomatic of fevers and hæmorrhagic disease, food should be given earlier.

Hæmathidrosis or **Hæmatidrosis.**—Cutaneous hæmorrhage without any apparent lesion of the skin; bloody sweat; hæmidrosis. *See* Skin, Diseases of Sweat and Sebaceous Glands (*Sweat Glands, Hæmidrosis*).

Hæmatin. *See* Fæces (*Abnormalities, Blood*); Physiology, Blood (*Decomposition of Hæmoglobin*); Pigments of the Body and Excreta (*Hæmoglobin and its Derivatives*); Spectroscope in Medicine (*Absorption Spectra*).

Hæmatinics.—Drugs and substances which increase the amount of hæmoglobin in the blood when it is deficient, such as iron, arsenic, and good food. *See* Pharmacology.

Hæmatinuria. *See* Blackwater Fever (*Nomenclature*); Malaria; Purpura; Scurvy in Adults.

Hæmatobium. *See* Filariasis.

Hæmatoblast.—An immature or imperfect form of the red blood corpuscles.

Hæmatocele. *See* Ectopic Gestation (*Intraperitoneal Hæmatocele*); Pelvis, Hæmatocele and Hæmatoma; Scrotum and Testicle, Diseases of (*Hæmatocele in the Scrotum*).

Hæmatochyluria.—The presence of blood and chyle in the urine. *See* Filariasis (*Filaria Bancrofti, Pathogenic Effects, Hæmatochyluria*).

Hæmatocolpos.—The retention of menstrual blood in an imperfectly developed (atresic) vagina, or in one half of a double vagina (*lateral hæmatocolpos*). *See* Generation, Female Organs, Arrested Developments.

Hæmatocrit.—An instrument for measuring the volume of blood corpuscles present in a fixed quantity of blood. *See* Blood (*Clinical Examination, Hedin's Hæmatocrit*).

Hæmatocytolysis.—The process of the breaking up of blood corpuscles.

Hæmatocytometer. *See* Blood (*Enumeration of Corpuscles*); Hæmacytometer; Hæmocytometer.

Hæmatogen. *See* Chlorosis (*Treatment, Special*).

Hæmatogenesis.—The development of the blood corpuscles. *See* Blood (*Developmental History*).

Hæmatoidin. *See* Expectoration (*Microscopical Examination, Crystals*); Liver, Diseases of (*Pigmentation*); Physiology, Blood

(*Decomposition of Hæmoglobin*); Physiology, Food and Digestion (*Bile, Pigments*); Pigments of the Body and Excreta.

Hæmatology.—The science of the blood both in its normal state and in pathological conditions.

Hæmatoma. *See* Aneurysm (*Intramural Hæmatoma, Arterial Hæmatoma*); Aneurysm (*Subclavian, Hæmatomata*); Axilla (*Injuries, Hæmatoma*); Broad Ligament, Diseases of (*Hæmatoma*); Bursæ, Injuries and Diseases of (*Injuries*); Contusions (*Morbid Anatomy, Hæmatoma*); Head (*Injuries of Scalp, Cephalhæmatoma*); Labour, Diagnosis and Mechanism (*Podalic Lies, Hæmatoma of Infant's Sterno-Mastoid*); Labour, Post-partum Hæmorrhage (*Traumatic, Hæmatoma Vaginæ*); Labour, Injuries to Generative Organs (*External Organs, Vulvar Hæmatoma*); Mammary Gland, Diseases of (*Mastitis*); Meninges of the Cerebrum (*Anatomy, Hæmatoma of the Dura Mater*); Muscles, Diseases of the (*Rupture*); Neck, Region of (*Tumours, Sterno-Mastoid Hæmatoma*); New-born Infant (*Cephalhæmatoma, Hæmatoma of Sterno-Mastoid Muscle*); Orbit, Diseases of (*Injuries of Soft Parts*); Ovaries, Diseases (*Complications of Ovariotomy, Hæmatoma*); Pelvis, Hæmatocele and Hæmatoma; Peritoneum, Tumours of (*Hæmatomata*); Pharynx, Examination (*Hæmorrhages*); Puerperium, Pathology (*Puerperal Hæmatoma*); Purpura (*Cutaneous Hæmorrhage*); Scrotum and Testicle, Diseases (*Hæmatoma of Cord*); Vagina, Disorders of (*Injuries, Hæmatoma*).

Hæmatoma Auris.

See also Ear, External, Diseases of (*Auricle, Hæmatoma*); Insanity, Pathology of (*Pathological Anatomy*).

Conditions of Occurrence.—Hæmatoma auris, otherwise known as othæmatoma, or the insane ear, occurs in its typical form in persons of unsound mind. A few instances have been recorded of the occurrence of an apparently similar condition unassociated with insanity, but in all probability either these cases are purely traumatic in origin, and therefore essentially dissimilar, or the patients are of degenerate habits, and mentally not far removed from the overtly insane. The broad fact remains that it is in lunatic asylums that the great majority of such cases are found. Even among the insane, however, only a certain small proportion suffer, estimated by Lennox Browne at 2·24 per cent, by Campbell at 2·23 per cent, and by Langdon Down as high as 3·6 per cent of male congenital idiots. It may be noted that these figures over-estimate rather than under-estimate the average frequency in most modern asylums. The number of men affected is relatively greater than the number of women. A somewhat peculiar and inexplicable fact is that the left ear is much more commonly involved than the right. Moreover, when both ears are implicated, as they are in about one-third of the total number of cases, it is the left which is usually first affected.

There is no particular form of insanity exclusively associated with othæmatoma. It has been found in all the more common varieties, but there is some difference of opinion concerning the types with which it is especially correlated. Clouston states that it is very common in general paralysis, and is sometimes seen in bad cases of mania of the chronic variety, sometimes in chronic epileptics, occasionally in agitated and convulsive melancholia, and rarely in dementia. Lennox Browne found that it occurred for the most part in patients subject to attacks of a violent and paroxysmal character. Out of thirty-two cases collected by him, seventeen suffered from mania acute or chronic, eight from general paralysis, five from epilepsy, and only two from dementia (not epileptic). Langdon Down, in noting its frequency in male congenital idiots, states that it is rare in female idiots and in accidental idiocy, that it may be found in idiocy arising from developmental causes, but almost never when the cause is operative in the early days of post-uterine life. The majority of idiots whom he observed to be subject to othæmatomata were also epileptic, and in all the element of excitement was excessive.

Clinical Features.—Othæmatoma of the insane first appears as a swelling in the external ear, and is almost invariably limited to the cartilaginous portion. It starts most commonly in the helix, and may then be bounded by the line of the anti-helix, or may extend more widely. Less frequently it begins elsewhere, as in the concha or fossa triangularis, and only very rarely in the external auditory meatus. In its earliest stage there occurs a small, somewhat red or livid, tense, cystic swelling which tends to increase more or less rapidly, until in the course of a few days it attains the size of a hazel nut or a hen's egg. The surface may be quite smooth, or may retain traces of the ridges in the fossa of the helix. Some irritation and discomfort are usually present, but seldom actual pain, and there are no subjective auditory symptoms such as deafness or tinnitus unless the external meatus is implicated. If proper treatment be withheld the cyst continues to enlarge, and may ultimately rupture, discharging

a bloody or gelatinous fluid. After a week or two, unless the process is interrupted by fresh hæmorrhages, the cyst contents begin to be absorbed, the cyst wall undergoes shrinkage, and ultimately the auricle becomes greatly atrophied, puckered, and permanently distorted. In exceptional cases, quite apart from any surgical interference, there may occur accidental inoculation with pyogenic microbes, suppuration sets in, and the cyst contents become purulent. Such a condition may, though rarely under antiseptic treatment, lead to extensive necrosis and gangrene of the external ear.

It is worthy of note that the prompt application of blistering fluid to the site of the swelling may, and very often does, not only arrest its further course, but greatly diminishes the amount of the subsequent deformity.

Morbid Anatomy.—Examination of a recent case of othæmatoma of the insane reveals the presence of an irregular cystic cavity situate usually between the anterior perichondrium and the cartilage of the pinna, as if these structures had been torn asunder by an effusion of blood. The cyst contents present great diversity of appearance, being sometimes dark red in colour and watery in consistence, sometimes pale yellow, translucent, and gelatinous. The effusion for the most part consists of blood in various stages of coagulation and decolorisation. The wall of the cyst shows an irregular lining of granulation tissue with newly-formed blood-vessels, which are often extensively degenerated. The adjacent cartilage shows numerous patches of degeneration which are described below, and which in all probability represent the essential preliminary lesion. In all ordinary cases which have not undergone accidental inoculation there is an entire absence of micro-organisms both from the cyst cavity and from the diseased cartilage.

As the condition persists, an increased formation of granulation tissue lines the interior of the cyst wall, organises and absorbs the effusion, and ultimately, by its further development into dense fibrous tissue, produces the extreme permanent contraction and distortion so characteristic of long-standing untreated cases.

Etiology.—Widely divergent views have been expressed regarding the causation and mode of origin of this condition, but only a brief outline of the more important theories need be given here. Most observers would probably concur in the statement that there is in the first instance some local predisposing condition under the influence of which the insane are rendered more prone than those in full mental health, and in the second place some local determining factor to the operation of which the actual onset is due.

The nature of the local predisposing cause was first suggested in 1848 by Fischer, who discovered the presence of cysts in the ear cartilages of the insane, and ascribed the occurrence of hæmatomata to primary hæmorrhage into them. Subsequently Virchow in 1863, Pareidt in 1864, L. Meyer in 1865, and Tischkow in 1891, confirmed and elaborated these earlier observations. More recently Ford Robertson in 1896 completed an extensive and careful examination of the ear cartilages both in the sane and in the insane, and found that in each series of cases there were present degenerative changes similar in kind but varying greatly in degree. The earliest evidence of degeneration is observed in the cartilage cells, which throughout areas of varying size became vacuolated and later disintegrated. The yellow elastic fibres in the same area are broken up into minute droplets and finally disappear. The central portion of the degenerated patch, more especially if large, tends to undergo liquefaction, thus leading to the formation of a small cyst. Vascular granulation tissue then replaces the degenerated patch of cartilage or lines the wall of the small cyst. In fifty cartilages from presumably sane patients, Ford Robertson found that no fewer than forty-eight showed some degenerative change, but he states that in the majority of these it consisted merely in the loss of the elastic fibres, and in slight degeneration of the cartilage cells in exceedingly minute areas. Granulation tissue replaced small areas of cartilage in eleven cases; but only in eight cases had the process advanced to the formation of a cyst, and in no case had vascularisation of the cyst wall occurred. In fifty cartilages from the insane there were as before only two cases in which degenerative changes were entirely absent, but in the number of areas affected and in the extent of tissue involved there was a contrast of the most marked kind. Cyst formation had occurred in no fewer than thirty cases, and in eight of these the cyst wall had become vascularised. There was, in short, abundant evidence of profound nutritional changes of a degenerative nature in the ear cartilages of the insane as compared with those of the mentally sound. Moreover, the young vessels in the newly-formed granulation tissue were themselves extensively degenerated, and thereby rendered unduly liable to rupture.

Many other explanations have been offered of the predisposition of the insane to othæmatoma, but these are rather tentative suggestions than statements of actual fact. Thus Alexander Robertson considered that a local vascular engorgement produced by disturbance of the cervical sympathetic system was the essential predisposing cause, and in this hypothesis he was supported by Pietersen and others. An obvious comment is that in exophthalmic goitre—a disease in which extreme disturbance of the cervical sympathetic system is undoubtedly present—there is no special predisposition to othæmatoma. Even such conditions as a blood dyscrasia, a lesion of the restiform bodies, etc., have been alleged to be the chief causal agents.

In all probability the actual effusion of blood from the diseased new vessels in the degenerative ear cartilage is determined by some insignificant traumatism. A slight accidental blow, or even the mere friction of the ear between the head and the pillow in a restless patient would suffice. It is quite unnecessary to assume that there has invariably been rough or careless treatment or a severe local contusion, and it is quite erroneous to maintain, as has been done by several writers, that all cases of othæmatoma are purely traumatic in origin.

Onset apart from Insanity.—Cases of hæmatoma auris in persons who are not under treatment for mental disease do undoubtedly occur, and may be included in one of two categories. If the subjects are in full mental health with undegenerated ear cartilages, the condition is unknown apart from severe local injury, *e.g.* fracture of the cartilage and laceration of healthy vessels. It has been known to occur at football, boxing, wrestling, or in other circumstances where the ears are exposed to considerable violence, but even then it is exceedingly rare. Moreover, the frequency with which some children receive a violent box on the ear without any damage of this nature, shows that, when the vessels and cartilages are healthy, hæmorrhage is not readily produced. Another class of patients, however, though not confined in asylums for the insane, includes those of degenerate mental powers, alcoholic habits, etc. From the researches of Ford Robertson on the ear cartilages of the presumably sane it is permissible to infer that in such cases considerable degeneration of the cartilage is also present. In other words, these represent a group of transitional cases in which the degeneration is not so advanced as it is in the insane, but yet sufficiently marked to allow hæmorrhage to occur from an injury of only moderate severity. The literature of the subject abounds with cases which support this view.

Relation to Microbic Infection.—The earliest reference to the infective origin of othæmatoma was made in 1846 by Leubuscher, who applied the term erysipelas auriculæ to the condition. Subsequently, from 1892 to 1896, Pellizzi published the results of a bacteriological research, from which he concluded that the ordinary hæmatoma auris of the insane is due to a streptococcus indistinguishable from the streptococcus pyogenes vel erysipelatis. Vassale, quoted by Pellizzi, failed to find any organism in one case examined by him. In 1894 Goodall stated that out of seven cases five showed the presence of the pyogenic staphylococci, both aureus and albus, while two were absolutely sterile. The attention of the writer was directed to this subject in 1895, and two typical cases of othæmatoma were examined. In each the cyst contents were at first perfectly free from micro-organisms, but in one case accidental inoculation subsequently occurred, the cyst contents became purulent, and then, and not till then, streptococci were abundantly present. Pieces of ear cartilage showing the early degenerative changes which precede the onset of an actual hæmatoma were also investigated, but no bacteria were detected.

The organisms so frequently present must be either the cause or an accident of the condition. In the former case, either they must have the power of producing those early degenerative changes in the cartilage which precede the formation of the hæmatoma, or they must by their presence determine the occurrence of the hæmatoma from a degenerated patch already present, or from healthy cartilage. There is, however, absolutely no proof that micro-organisms are at all related to simple cartilage degeneration. Hence, if the presence of micro-organisms is not to be considered purely accidental, it must be shown that they are the direct cause of the hæmatoma. This is the position for which Pellizzi contends, and which Goodall is inclined to support, but it is quite untenable, for the following reasons:—(1) The cases quoted by Pellizzi, Goodall, and the writer, show that no single organism can be regarded as the specific cause, since at least three different varieties have been found. (2) The results of Vassale, Goodall, and the writer, further show that organisms are by no means constantly present, but that there is a considerable proportion of cases which, when examined with all due care, give purely negative results. (3) By inoculation of pure cultures Pellizzi has consistently failed to reproduce the condition, causing simply inflammation and suppuration in varying degrees of severity. (4) The organisms hitherto discovered are all identical with well-known pyogenic forms which have never been found to produce any condition analogous to hæmatoma auris.

The conclusion, therefore, to which the evidence most strongly points is, that the relation of micro-organisms to othæmatoma is purely accidental. It is not the presence of organisms that determines the formation of the hæmatoma, but, on the contrary, it is the hæmatoma that affords a subsequent nidus for organisms. Nor is it difficult to understand how such access may be obtained. Any subcutaneous effusion is more or less liable to become inoculated, and, in the insane, this liability is considerably increased, more especially when such an exposed part as the ear is affected. For, owing to the carelessness, or restlessness, or actual violence of the patient, or owing to his fingering the damaged part, there may readily be produced an abrasion which may become the channel of infection.

Prognosis.—The result to the auricle affected depends chiefly on the promptitude with which suitable treatment is applied. If the condition

be permitted to develop to its full extent without any remedial measures being undertaken, the subsequent deformity is usually extreme; whereas the early application of blistering fluid usually succeeds in arresting the progress of the effusion, and in very greatly diminishing the amount of permanent deformity.

The prognostic value of othæmatoma as regards the progress of the mental malady is important. Its presence is indicative of widespread and profound nutritional changes, and a corresponding extent and degree of cerebral degeneration. The prospect of ultimate recovery is, therefore, very slight. Clouston states that he has "seen only four or five cases perfectly recover out of over eighty who had fully developed hæmatoma auris, and four others who made partial recoveries after slight threatenings of hæmatoma auris which might not have developed fully or were stopped by blistering fluid."

Treatment.—The most effective means of treatment is that first suggested by Dr. Hearder, and indicated in the preceding section. It consists in the application of blistering fluid to the auricular surfaces on the earliest signs of any swelling from the commencing hæmatoma. Its success depends upon the promptitude with which the fluid is painted on, and the failure of the method is usually proportionate to the delay. If, as sometimes happens, the cyst becomes accidentally inoculated with pyogenic organisms, it must be freely incised, and the purulent contents washed out with antiseptic solutions. Except in these circumstances, however, surgical interference should be avoided as tending more to aggravate than to improve the condition.

Hæmatometra.—Distension of the uterus with blood, usually retained menstrual blood. *See* Generation, Female Organs (*Arrested Developments*); Pregnancy, Diagnosis (*Retention of Menstrual Fluid*); Uterus, Malformations of (*Atresia of Vaginal Orifice*).

Hæmatomonas Evansii.—The so-called *surra* parasite, a microbe believed to be the cause of surra, a disease affecting horses, camels, etc., and occurring in India.

Hæmatomyelia.—Hæmorrhage into the substance of the spinal cord. *See* Spinal Cord, Medical (*Vascular Lesions, Hæmorrhage*).

Hæmatonephrosis.—Hæmorrhage into the substance of the kidney, cystic or simple.

Hæmatonosus.—A blood-disease, also a cutaneous malady characterised by vascular disturbances in the tissues of the skin (*e.g.* eczema).

Hæmatopericardium.—The effusion of blood into the pericardial sac.

Hæmatophilia. *See* Hæmophilia, although *hæmatophilia* is perhaps the more correct form.

Hæmatopoiesis.—Hæmatogenesis or blood formation. *See* Physiology, Blood (*Source of Blood Constituents*).

Hæmatoporphyrinuria.

See also Physiology, Blood (*Decomposition of Hæmoglobin, Urine, Pigments*); Pigments of the Body and Excreta (*Hæmatoporphyrin*); Rheumatism, Acute (*Symptoms, Urine*); Spectroscope in Medicine; Urine, Pathological Changes in (*Colour, Pigments*).

The condition known as hæmatoporphyrinuria has attracted considerable attention of recent years. It is characterised by the passage of urine of a dark red colour, like that of port wine, and which, although free from hæmoglobin, contains a considerable amount of that derivative of the blood pigment which is known as hæmatoporphyrin.

This employment of the term, although very convenient, is open to certain objections. In the first place, hæmatoporphyrin is present in minute quantity in normal human urine, and under various morbid conditions the amount present is notably increased, so that it is sometimes possible for a trained eye to detect some of its characteristic absorption bands when a sufficiently deep layer of the urine is examined with the spectroscope. However, under ordinary conditions the quantity present is too small to have any material influence upon the colour of the liquid.

Secondly, in the condition known as hæmatoporphyrinuria there is not merely an exaggeration of the increased hæmatoporphyrin excretion, which is so common a phenomenon in disease, but rather a profound disturbance of pigment metabolism of which this is only one of the evidences. These dark red urines owe only a small part of their abnormal colour to the hæmatoporpyhrin which they contain, as Hammarsten was the first to point out, and their tint is mainly due to the presence of other abnormal pigments of which we as yet know very little. Moreover, even the known pigments which they contain are apt to exhibit certain peculiarities in their behaviour to solvents, and in other respects.

The frequent dependence of such hæmatoporphyrinuria upon the administration of sulphonal is no longer open to doubt, and the allied drugs trional and tetronal appear to have a similar action, only in a lesser degree. In

the great majority of the recorded cases the urinary change has formed one of a group of toxic symptoms following upon the taking of sulphonal for a longer or shorter period. However, of the many patients who nowadays take this hypnotic, only very few ever exhibit such symptoms, and it is a remarkable fact that with few exceptions those who so suffer are women. The few recorded cases in males have been for the most part of a mild character.

Patients taking sulphonal do not, under ordinary circumstances, excrete unusual amounts of the pigment in question, and those who develop hæmatoporphyrinuria have often taken sulphonal nightly, for weeks or months, with impunity. Sometimes the toxic symptoms only make their appearance after the drug has been discontinued, and in one of Hammarsten's cases the interval was as long as nine days. Sometimes, again, they have followed the administration of very few doses.

Of the associated symptoms, those most frequently observed are vomiting, constipation or diarrhœa, abdominal pain, and paresis or paralysis of limbs. In severe cases the patients pass into a state of collapse, with cyanosis and coldness of the extremities, and death occurs at an early period. In favourable cases, on the other hand, the attendant symptoms quickly subside, and the urine gradually regains its natural colour.

The changes found post-mortem in fatal cases have not been very characteristic. Submucous hæmorrhages have sometimes been present in the stomach and intestines, but are by no means constant. The liver usually shows conspicuous fatty degeneration, and granular or necrotic changes in the epithelium of the renal glomeruli and tubules bear witness to a toxic nephritis.

It appears equally certain that hæmatoporphyrinuria occasionally, but rarely, occurs apart from the administration of sulphonal or allied drugs. In these days of self-medication it is not always easy to exclude their use in a given case, but there are examples on record in which this could be definitely excluded, and some of these occurred before the introduction of sulphonal.

As a rule these cases have run a favourable course, but in two recorded by Ranking and Pardington, one of which proved fatal, other symptoms were present similar to those observed in sulphonal cases.

Neusser met with hæmatoporphyrinuria in association with phthisis pulmonalis in one case, and with pleurisy with effusion in another; MacMunn in a case of exophthalmic goitre; MacCall Anderson saw repeated attacks in association with recurrences of hydroa æstivalis. Nebelthau has recorded its occurrence in a case of congenital syphilis, and Sobernheim in the case of a boy admitted to hospital as suffering from typhoid fever. The only case of this kind which I have had the opportunity of watching was that of a woman admitted to hospital for hæmatemesis, and who was under the care of Dr. Calvert. Although no evidence of the taking of sulphonal could be elicited by careful inquiry, the characters of the urine were very similar to those met with in the sulphonal cases, the dark colour being mainly due to abnormal pigments other than hæmatoporphyrin.

In cases of hæmatoporphyrinuria the colour of the urine usually resembles that of tawny port-wine, but it may be so dark as to approach to black. As a rule it is free from albumin, but by the aid of the centrifuge a few tube-casts may usually be detected. The reaction is usually acid, often strongly so, and the urine may usually be kept for a long time without decomposition. In some sulphonal specimens, which Hopkins and I examined, there was no increased excretion of iron.

Direct spectroscopic examination does not usually yield such satisfactory results as might be expected considering the amount of hæmatoporphyrin present. The other abnormal pigments cause much general absorption of the blue end of the spectrum, and often greatly interfere with the view of the absorption bands. However, the narrow band in red of alkaline hæmatoporphyrin can usually be made out (Fig. 2).

Frequently the bulk and sometimes the whole of the hæmatoporphyrin present is in the form in which it shows what is known as the "metallic" spectrum (Fig. 3), consisting of two dark bands resembling those of oxyhæmoglobin, and this introduces a further difficulty in its detection by direct spectroscopic examination. The addition of hydrochloric acid causes the acid spectrum (Fig. 1) to appear, but this spectrum is never seen, even with strongly acid urine, until a mineral acid is added.

More satisfactory results are usually obtained by extraction with acetic ether after the addition of acetic acid. By this means a solution of hæmatoporphyrin is obtained comparatively free from the associated pigments, but occasionally it refuses to be taken up even by this solvent.

The method of precipitation of the pigment with the earthy phosphates, by adding a 10 per cent solution of sodium hydrate (20 c.c. for each 100 c.c. of urine), and extraction from the washed precipitate with acid alcohol, although it serves admirably for the detection of hæmatoporphyrin in normal and ordinary morbid urines, does not usually give good results in these cases. Salkowski's method of precipitation with equal volumes of 10 per cent solution of barium chloride and baryta water is certain in its action, but has the disadvantage that the acid alcoholic extract obtained from the precipitate contains the other abnormal pig-

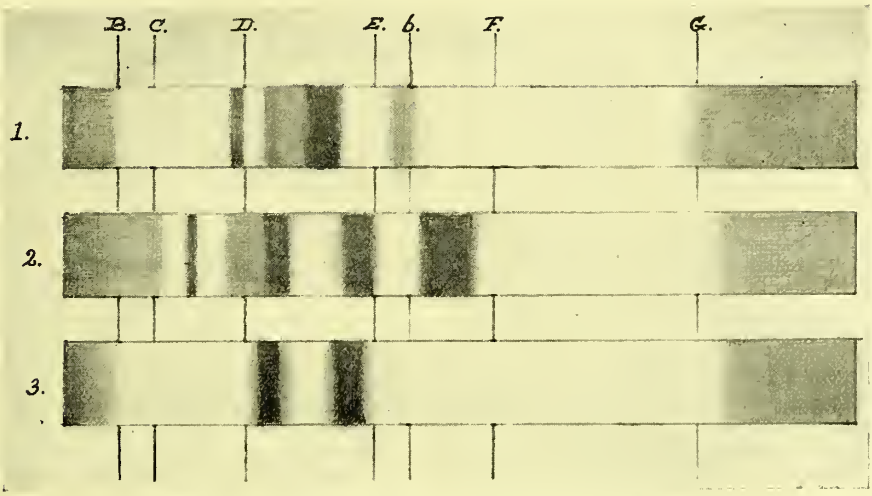
ments. Nebelthau succeeded in precipitating the hæmatoporphyrin by adding glacial acetic acid to the urine (5 c.c. acid to 100 c.c.) and allowing it to stand for 24-40 hours.

Of the processes which are at work in the production of hæmatoporphyrinuria, we know very little as yet. Stokvis found that when sulphonal was administered to rabbits hæmatoporphyrin appeared in their urine. Kast and Weiss were not convinced that the pigment excreted under these circumstances was really of this nature, but Stokvis's observation has recently been confirmed by Otto Neubauer. Stokvis also found submucous hæmorrhages in the stomach of rabbits poisoned with sulphonal, which gave the spectrum of acid hæmatoporphyrin, and he was inclined to attribute the hæmatoporphyrinuria to the action of the gastric juice aided by sulphonal upon the blood so extravasated. However, the records of post-mortem examinations in fatal cases of this condition lend little support to this theory, for although such hæmorrhagic areas have sometimes been found, their presence is by no means constant.

Blood counts made in a number of cases have failed to show any very conspicuous or progressive diminution of red corpuscles in association with the hæmatoporphyrinuria, and the evidence available points rather to a perversion of the ordinary chemical processes for the disposal of effete blood pigment than to excessive hæmolysis as the cause of the phenomenon.

It should be mentioned that in some instances the administration of trional has been followed by the passage of dark urine rich in urobilin, but without any marked excess of hæmatoporphyrin. Urobilin is also usually present in considerable amount in the ordinary dark red urines.

As to treatment little can at present be said. In sulphonal cases the immediate discontinuance of the drug is clearly indicated. Franz Müller attached much value to treatment with alkalies in his case, and a trial should certainly be given to this plan, which receives support from some experiments of Stokvis upon rabbits. Two cases which have come under my observation, in which alkalies were given, ran a favourable course, but neither was orignally of a severe character.



Spectra of Hæmatoporphyrin.—1. Acid; 2. Alkaline; 3. Metallic.

Hæmatopota pluvialis. — The clegg; a parasite.

Hæmatorrhachis.—An effusion of blood around or between the spinal membranes.

Hæmatosalpinx. *See* Fallopian Tubes; Menstruation and its Disorders (*Varieties and Causes of Dysmenorrhœa*); Uterus, Malformations of (*Retention of Menstrual Products, Atresia of Vagina*).

Hæmatoscope.—An instrument for observing the spectroscopic characters of the blood; hæmatospectroscope.

Hæmatothorax.—An effusion of blood into the pleural cavity or cavities.

Hæmatotrachelos. — Retention of menstrual blood in the cervix uteri.

Hæmatoxyli Lignum.—The heart wood of a tree (*Hæmatoxylon campechianum*) belonging to the Leguminosæ, and known as Logwood; in Pharmacy, it is met with as reddish chips, with a pleasant odour and a sweetish and astringent taste; it contains *tannic acid* and *hæmatoxylin* ($C_{16}H_{14}O_6$); it is used as an astringent in diarrhœa, but it is incompatible with lime water; the official preparation—the Decoctum Hæmatoxyli—is given in doses of $\frac{1}{2}$ to 2 fl. oz. *See also* Astringents.

Hæmatozoa. — Animal parasites of various kinds which live on the blood or the blood-vessels. *See* Filariasis ; Malaria ; etc.

Hæmaturia. *See also* Abdomen, Injuries of (*Kidney*) ; Abdominal Aneurysm (*Pressure Symptoms*) ; Bladder, Injuries and Diseases (*Calculus, Symptoms ; Tumours, Symptoms*) ; Children, Clinical Examination of (*Urine*) ; Cholera, Epidemic (*Symptoms*) ; Circumcision (*Complications of Phymosis*) ; Heart, Myocardium and Endocardium (*Symptomatology, Kidneys*) ; Hysteria (*Disorders of Urinary System*) ; Kidney, Surgical Affections of (*Injuries, Symptoms*) ; Kidney, Surgical Affections (*Tuberculosis, etc., Symptomatology*) ; Kidney, Surgical Affections (*Stone in the Ureter*) ; Mumps (*Complications*) ; Rheumatism, Acute (*Renal Affections*) ; Rheumatism in Children (*Purpura and Hæmaturia*) ; Scurvy, Infantile (*Clinical Features, Hæmaturia*) ; Urine, Pathological Changes in.

According to its etymological sense the term should be restricted to cases in which blood is effused from the vessels of the kidneys, ureters, or bladder, and discharged along with the urine, excluding from the definition urethral hæmorrhage, in which the blood escapes by drops or flows in a continuous stream from the orifice of the urethra, and which is not properly a mictus cruentus.

The appearance of blood in the urine during micturition is a symptom of a large number of different lesions, and, as seen in surgical practice, may have its origin in any of the divisions of the urinary tract. Hæmaturia may be due to (*a*) lesions of the renal parenchyma or the pelvis ; (*b*) disease of the ureters ; (*c*) disease of the bladder ; (*d*) disease of the prostate ; (*e*) disease of the urethra ; and (*f*) disease of the testicles.

The pathological conditions which are associated with the appearance of blood in the urine are therefore very various and numerous. The great majority come under the cognisance of the surgeon, but there are some which belong strictly to medical practice. For example, hæmaturia may be an accompaniment of hæmophilia, septicæmia, typhus, enteric, or malarial fevers, small-pox, scurvy, or purpura ; or it may be the direct consequence of poisonous agents, such as cantharides, alcohol, turpentine, phosphorus, and arsenic.

There is an intermittent form of hæmaturia which has been described under the term "malarial hæmaturia," more common in men than in women, and differs from ordinary "paroxysmal hæmaturia" in the greater regularity of the attacks, in that it is met with in certain districts only, and in that it is associated with the presence of malarial organisms in the blood.

What is called "endemic hæmaturia" depends upon the presence of a parasite, the *Bilharzia hæmatobia*. This form of the disease is met with in Egypt, Natal, Cape Colony, Mauritius, and in Brazil, and a somewhat similar hæmaturia prevails in India, due to the presence of the *Filaria sanguinis hominis*. These conditions only require to be mentioned, as they are rarely met with by practitioners at home. (*See* "Parasites.")

Tests. — Tests for blood in urine may be divided into three classes :—

(*a*) *Microscopic.*—When the urine is allowed to stand the corpuscles and colouring matter may fall as a deposit, when they are easily recognised by the microscopic examination. This is most readily seen when the urine is acid or neutral ; but when it is alkaline the colouring matter is liable to be dissolved out of the corpuscles, and these by imbibition of fluid become distended and appear as almost colourless spheres instead of flat discs. Their characteristic appearance is therefore lost. Occasionally the edges may become serrated from shrivelling of the corpuscles.

(*b*) *Spectroscopic* examination affords a very delicate test. Blood even in very minute quantities gives the characteristic absorption bands of hæmoglobin in the yellow and green between the D and E lines.

(*c*) *Chemical* tests may be also employed. (1) The addition of carbolic acid to a urine containing blood causes coagulation of any albumin which may be present, and also changes the colour of the fluid to a peculiar reddish tinge. (2) By boiling the urine with caustic soda and allowing it to stand, a brick-red precipitate is thrown down. (3) When a little glacial acetic acid and a crystal of common salt are added to urine containing blood, Teichmann's crystals are deposited. (4) When a couple of drops of tincture of guaiacum and half a drachm of "ozonic ether" are added to a drachm of the urine in a test-tube and the whole shaken, the ether dissolves the resin which has been precipitated, and after a few seconds carries it to the surface of the urine. At the line of union between the "ozonic ether" and the fluid lying underneath it, if blood be present, a blue coloration appears.

Fallacies.—Red urine is sometimes passed by patients who have taken suphonal in large doses or for a long period. The excretion may also be rendered red or black from the admixture of various pigments derived from articles of diet or medicine. Rhubarb, beetroot, strawberries, sorrel, prickly pear, and logwood may impart a red colour. Senna, salicylic acid, and carbolic acid may also cause coloration.

In microscopic examinations, globular vegetable spores may simulate blood corpuscles. In using the spectroscope it must be remembered that logwood gives a spectrum very similar to hæmoglobin, and in employing the guaiacum test

it must be borne in mind that saliva gives the same reaction as blood.

Differential Diagnosis of the Source of the Hæmorrhage.—The methods employed may be classified as follows :—

Firstly, by observing (*a*) the physical characters of the urine and of the blood-clot; (*b*) the admixture of other deposits with the blood; (*c*) the time at which the blood appears in the stream; (*d*) the frequency and duration of attacks; and (*e*) the effects of movements and exercise, or of complete rest, in the course of an attack. Secondly, by collecting the urine separately from the two ureters, or by observing blood escaping from the orifice of a ureter. Thirdly, by carefully considering all the other accompanying objective and subjective phenomena.

I. (*a*) *Physical Examination of the Urine and the Circumstances connected with the Presence of Blood in the Evacuation.*—The following questions must be inquired into and answered :—What is the colour of the blood? Is it intimately mixed with the urine, and is the whole stream equally colourised? Is the quantity of blood augmented by exercise or diminished by rest? Have clots been seen, and what is their form? Are there tube-casts or blood-casts to be discovered, or are there other deposits found in the urine? Are the attacks frequently repeated and of short duration, or are they protracted?

The colour imparted to the urine by the addition of blood varies greatly in intensity and shade—from a pale rose colour to a bright red, like red currant syrup mixed in water, to a dark red, to a colour simulating porter, or to a brown coloration not unlike coffee. As a general rule, however, it may be stated that the nearer the source of hæmorrhage is to the external orifice of the urethra the less is the blood altered in appearance.

When the hæmaturia is of *vesical origin*, the first quantity of urine passed may present a normal appearance, especially if the patient has been for some time at rest in a recumbent posture, and the urine is passed while the patient is in bed; but in such circumstances, as the bladder empties itself, the urine gradually becomes more and more tinged, till finally the last drops evacuated may be almost pure blood. This is to be especially noted in cases of tumour of the bladder and of vesical calculus, also in passive hyperæmia of the bladder. As a general rule, in hæmorrhage from the bladder the urine is mixed with a considerable quantity of mucus, and is of a bright florid colour; but, if long pent up in the viscus, it may assume a dark colour dependent upon the reaction of the urine, which in such cases is usually alkaline. When due to vesical calculus the hæmorrhage is increased by exertion, and in cases of tumour of the bladder the bleeding is always worse after an attempt has been made to examine the bladder by instruments.

When the blood is from the *kidneys* or the *ureters* it is generally intimately mixed with the urine, to which it imparts a dark smoky colour, unless when the blood issues in considerable quantity from the pelvis of the kidney, and flows rapidly along the ureter, distends the bladder, and escapes without undergoing much change.

Blood having escaped from the vessels, its fibrous constituents may coagulate into firm clots, which during their formation may entangle some of the histological elements of the structures in which they lie, or by their firm appearance, form, or bulk may indicate the source of the hæmorrhage. Clots are seldom noticed in the urine unless in cases of abundant hæmorrhage; they may be few in number or numerous; generally they are soft in consistence, and when small in size they are easily dissolved in the urine, and consequently, if not looked for immediately after micturition, they may be lost. The form of the clot may be ovoid, or may be long and worm-shaped, or may resemble in appearance a well-gorged leech. As to colour they are generally dark red, but may vary considerably, sometimes being black, bright red, or they may become partly or wholly discoloured—spotted red in a greyish background, or entirely grey. When the clot is large the hæmorrhage is usually, but not invariably, from the bladder; the clots may be so huge that they cannot pass along the urethra without being broken up either by the repeated contraction of the bladder or by means of instruments. Rounded clots corresponding to the diameter of the ureter may escape, or leech-like ovoid casts may be taken of the first part of the urethra, or long bougie-like coagula from its anterior part. While the presence of considerable clots is usually, though not always, conclusively against the idea of the hæmorrhage being from the secreting substance of the kidney, the absence of clots visible to the eye proves nothing. If the passage of a long worm-shaped blood-clot is preceded by a temporary cessation of the hæmaturia, and its escape followed by a recurrence of the bleeding, this proves almost to demonstration that the clot has been plugging one ureter; and if, in addition, the clot is of such a size as to support the idea of being a mould of the ureter, the evidence is conclusive as to the source of the hæmorrhage. In renal hæmaturia, minute coagula, casts of the uriniferous tubules, may be discovered in the urinary sediment by the microscope, and may give evidence by their form and shape whence they are derived. In this form of hæmaturia the number of blood corpuscles is no criterion of the quantity of albumin in the urine, whereas in hæmorrhage from the conducting or collecting portions of the urinary tract, provided there is no pus in

the urine, the number of red corpuscles or the amount of hæmoglobin may be accepted as the measure of the quantity of albumin contained in the excretion.

(*b*) *The Admixture of other Deposits with the Blood.*—In many cases of hæmaturia the only abnormal constituent in the urine is blood, while in other instances it is mixed with pus, mucus, tuberculous material, portions of tumours, or micro-organisms, the detection of which throws considerable light on the etiology of the malady. The mixture of the blood with deposits of another kind requires only to be mentioned here. The three most common are pus, mucus, and tuberculous débris. If the urine is placed in a conical glass and allowed to stand for a few hours, and the glass is then held up to the light, if pus is present in any great amount the deposits will be found in distinct layers like geological strata; the bottom of the glass is occupied by a yellowish deposit, which may be more or less blood-stained pus, or, in some cases of renal pyuria, the pus may carry down almost all the blood corpuscles, so as to leave an almost quite clear supernatant fluid. When mucus is present, on the other hand, not uncommonly the blood corpuscles may be thrown down first, forming a layer at the bottom of the vessel, and following upon this may be seen a layer of glairy gelatinous material, which may have a reddish tint, or may contain a multitude of minute bloody streaks which intersect it, and penetrate into all parts of this gelatinous layer. In such cases the hæmorrhage is usually due to some lesion in the bladder, and the presence of such an amount of mucus is generally indicative of a more or less acute vesical catarrh. The tuberculous deposit can only be distinguished by microscopic examination, by the cultivation of tuberculous bacilli, or by inoculation experiments. Beyond demonstrating the presence of blood-casts of the uriniferous tubules the microscope may reveal the existence of other elements of consequence in the deposit. For example, the character of the epithelium as derived from the renal parenchyma, from the pelvis, the ureter, or the bladder, may greatly assist one in diagnosis; or, on the other hand, should the urine contain small fragments of tumours, parasites, or bacteria, the hæmorrhage may be explained.

(*c*) *The Time at which the Blood appears in the Stream.*—In order to ascertain the precise moment at which the blood appears in the stream it is necessary for the surgeon to see the patient urinate, and to observe whether the coloration is equal during the whole continuance of micturition, or is more abundant at the beginning or at the end of the act. The appearance of blood at the beginning of micturition, the remainder of the flow being clear, may be an indication of two distinct conditions—the lesion is either in the prostate close to the neck of the bladder, or a malady or injury is present in the urethra. In some lesions of the prostate the blood may pass into the urethra and accumulate there; so also in lesions of the first portion of the urethra the blood may flow backwards into the bladder and mix with its contents. When the blood originates in the urethra and accumulates there, or when it escapes from the prostate and flows into the urethra, the hæmaturia is limited to the beginning of micturition; but if the quantity of blood is great, and flows backward into the bladder and mixes with its contents, then the whole of the urine becomes more or less coloured. Again, in a lesion at the neck of the bladder giving rise to hæmorrhage, the hæmaturia is not limited to the beginning of micturition, but blood is also observed to escape at the end of the act. This is easily explained. Between the acts of urination the blood accumulates in the urethra or close to its internal orifice, and is expelled with the first few drops of urine only, that following being clear; but again, before complete contraction of the bladder occurs, a fresh hæmorrhage is induced, which shows itself in the urine last ejected. When the hæmorrhage is due to injury or disease of the urethra, the nature of the lesion is generally clearly indicated by the circumstance that the blood is observed to escape quite independently of micturition; sometimes, indeed, blood is seen to flow from the meatus spontaneously. In cases of tumour of the bladder, and also in vesical calculus, the hæmorrhage is most profuse at the end of micturition.

(*d*) *The Frequency and Duration of the Attacks.*—While nothing absolutely definite can be ascertained by a close observation and study of the frequency and duration of the hæmaturia, still considerable help may be got in this way to aid in a diagnosis. For example, in many cases of renal hæmaturia the blood may suddenly appear and just as suddenly disappear, soon to be followed by a profuse recurrence; such sudden transformations are in some cases accompanied by the expulsion of long worm-shaped clots, and in such instances we may reasonably conclude that the sudden clearing of the urine has been due to the ureter being obstructed. The blood has coagulated within its lumen, and when the clot becomes displaced a fresh, and often apparently very profuse, escape of blood is observed. When hæmaturia comes on without being evidently provoked, we may generally surmise that the lesion giving rise to it is a serious one, although we may not be able to judge its site. In cases of tumour of the bladder the presence of blood is generally very persistent, without intervals, and of long duration, so that the patient may become very anæmic from loss of blood. Again, on the other hand, we meet with cases where hæmaturia more or less profuse has recurred at frequent intervals; but the duration of the attacks has been short, and the intervals

between them marked by complete absence of any blood in the urine. Sometimes there may be periods of relief extending over several weeks or even months, and usually any fresh recurrence of bleeding can be explained by excessive exercise or unwonted freedom of movement on the part of the patient. If these conditions are associated with the absence of vesical symptoms, the strong presumption is in favour of the hæmorrhage being renal in origin and against the hypothesis that the blood is flowing from an ulcerated surface; the total duration of the symptoms is also an important guide to the benign or malignant nature of the malady giving rise to it.

(*e*) *The Effects of Movement or Exercise or of Complete Rest in the Course of an Attack.*—When complete rest is taken, hæmaturia due to the presence of stone in the renal pelvis, in the bladder, or in the prostate, is generally more or less relieved; so also in hæmorrhage observed in cases of movable kidney, or in passive hyperæmia of the kidney resulting from pressure on the renal veins. In such cases it is repeatedly observed that the blood is most abundant in the urine at night when the patient has been taking active exercise during the preceding day, while at the same time the pain in the renal region or irritability of the bladder is increased. It must, however, be also borne in mind, in exceptional instances, that in hæmaturia from an abraded surface, as in carcinomatous or tuberculous ulceration, or in senile prostate, the bleeding occasionally may not be increased by exercise; but, as a rule, if the hæmaturia persists in spite of prolonged rest in bed, and especially when the bleeding is more abundant during the night than at other times, carcinomatous, sarcomatous, or tuberculous ulceration is to be looked for.

II. *By collecting the Urine separately from the two Ureters, or by observing Blood escaping from the Orifice of a Ureter.*—The cystoscope may be used both in men and women, and when properly employed the examination subjects the patient to very little risk. The sound is too frequently employed in cases of hæmaturia. It is only when the bleeding is the result of a vesical calculus, or an enlarged prostate, or an hypertrophy of the bladder, that any reliable information can be gained by the employment of the sound; and when the hæmaturia is due to other lesions, much harm may be done by the rough manipulation necessary to examine the bladder with that instrument. The educated use of the cystoscope, being more gentle, is less dangerous, and it is more valuable; but it should always be used with strictly aseptic precautions. In all cases, not only should the cystoscope itself be thoroughly sterilised, but the meatus and surrounding parts should also be carefully washed, and rendered as free from contaminating particles as possible; and after examination has been made the bladder should be carefully washed out with a fresh supply of suitable antiseptic solution. By making careful examination with the cystoscope (*vide* "Cystoscope," vol. ii. p. 264) it is usually easy to ascertain whether or not the blood is from the bladder; but in a few instances it is not safe to conclude from the fact of no lesion being observed in the bladder that the hæmaturia is therefore either renal or urethral in its origin. When, however, the blood is seen by the cystoscope flowing from the orifice of a ureter, or more rarely from both ureters, the observation is of the highest importance as indicating the source from which the blood comes.

Catheterisation of the Ureters.—Catheterisation of the ureter in the female has been much simplified by Kelly of Baltimore.[1] The essential feature of his method is that of the introduction of a straight speculum into the empty bladder. The walls of the viscus are slightly separated by the position assumed by the patient, the dilatation being such as to bring the orifices of the ureters into view when reflected light is thrown into the bladder by a forehead mirror. Those who desire to catheterise the ureters must carefully study their direction in the various parts of their course, and must always remember that gentle manipulation is of the highest importance.

Within the last few years two catheterising cystoscopes have been introduced, the one by Nitze, and the other by Casper. In catheterising the ureters it must be borne in mind that rough introduction of the catheter may of itself lead to hæmorrhage, and certainly this is a drawback to its use, especially when the catheterised ureter is the seat of inflammation. In the female the procedure is easily carried out, and the results are tolerably certain; but in the male, even when one succeeds in introducing the catheter into the ureter, it is difficult to say to what degree the results obtained are to be relied upon.

A point of considerable importance in diagnosis is the appearance of the orifice of the ureter as seen by the cystoscope.

III. *By carefully considering all the other Objective and Subjective Phenomena.* — The questions which have been considered up to the present give but a rough sketch; the detail requires to be carefully filled in before the picture is completed. In order to do this the practitioner must consider the most common sources of hæmaturia, and enumerate the various lesions of the kidney, the ureters, the bladder, the prostate, the urethra, and the testicles, which may give rise to the symptom under discussion. It is well always to conform to this rule in going over the etiological factors which have been indicated above.

[1] *Twentieth Century Practice of Medicine*, 1895, vol. i. p. 675.

Placing aside for the present the hæmaturia due to hydatid disease, the renal hæmorrhages of the greatest practical importance, from our present standpoint, are those arising from injury, renal calculus, tumours of the kidney, and tuberculous disease of that organ. Having determined that the hæmorrhage is not from the lower urinary tract, it remains to ascertain which of those four causes is to blame.

When the hæmorrhage is due to *renal calculus* it is usually small in amount, sometimes constantly present, but generally with more or less prolonged intervals, and commonly oft-repeated. In some instances, however, the hæmaturia is very slight, while in other cases it may be the only symptom. The bleeding is not closely related to pain or to the development of other symptoms, but as a rule it is increased by movements of the body. This, however, does not follow immediately, but a short interval may elapse—hours, or even days—between the exertion and the appearance of the blood in the urine. While the hæmaturia of renal calculus is more copious after exercise, rest in bed usually appreciably diminishes it. This peculiarity is most characteristic. The blood, it must be borne in mind, is derived from the renal pelvis and not from the parenchyma; consequently renal blood-casts are not found in the urine, and the quantity of albumin is fully explained by the presence of blood. When the urine is evacuated the blood is thoroughly mixed with it, but not so intimately as when the cause of the hæmaturia is structural disease of the kidney, and if the urine be allowed to stand for a few hours the blood corpuscles are readily precipitated and leave the supernatant urine clear. If free from blood the urine will also be found to be non-albuminous. The presence or absence of pus in the urine will be determined by the circumstance whether or not the calculus has induced inflammatory changes, and the existence of a swelling in the renal region will depend upon the amount of freedom for the escape of urine by the ureter.

Renal hæmorrhage from *tumour* is generally more profuse and less transient than from calculus, and in not a few cases it is so copious as to cause marked anæmia—a result seldom induced by calculous hæmaturia. It is often developed without any preceding pain. In calculous disease injury or exercise commonly provokes the bleeding, and therefore one finds the hæmorrhage more profuse during the day while the patient is moving about. The bleeding from tumours is, on the contrary, most likely to occur during the night, while the patient is at rest in a recumbent posture. The urinary deposit may assist one in the diagnosis. Concretions composed of oxalate of lime, uric acid, phosphates, or urates may indicate the character of the stone; while by carefully filtering the urine in cases of tumour, portions of the growth may be procured for microscopic examination. The presence of a persistent swelling in the renal region, associated with considerable hæmaturia, is of significance, and may be held as clearly indicating the presence of a neoplasm in the kidney. Exceptions to this rule have, however, been recorded by Ebstein, Hirtz, and Fleming. But while this is so, it must not be forgotten that the presence of a palpable new formation may for a considerable time be preceded by the presence of blood in the urine. Considerable distension of the pelvis may be produced by a calculus obstructing the ureter, and so lead to a swelling in the renal region, or the bulging in the loin may be caused by enlargement of the kidney from tuberculous disease; in neither of these conditions, however, does hæmaturia frequently occur. In the latter the very obstruction which causes an increase in bulk of the kidney prevents the hæmorrhage.

Although much difficulty is often experienced in distinguishing calculous hæmaturia from that caused by tumours, there is still greater care required in the diagnosis between the hæmorrhage of early tuberculous disease and that of renal calculus. The symptoms of the two latter conditions are sometimes identical. This is especially so when the patient fails to show any other evidence of tuberculous disease than that revealed by the renal lesion.

In *tuberculous disease* hæmaturia is frequently absent for long intervals, is seldom so severe as in stone, and is not increased by exercise. In both conditions pus may be mixed with the urine; but while in the latter concretions and gravel may be discovered, in the former careful and repeated search may demonstrate the presence of tuberculous bacilli. In the early stage of tuberculous disease of the kidney the quantity of albumin in the urine is generally in excess of that accounted for by the blood, and in the later stages, when pus appears in considerable quantity, the pus and blood are not so rapidly or so completely precipitated from the urine as in calculous pyelitis. The presence of phthisis pulmonalis, tuberculous disease of bones or joints of the testicle, the prostate, the vesiculæ seminales, the mesenteric glands, the intestine, or of the lower urinary tract, may give a clue to the cause of the hæmaturia.

Hæmautograph.—The tracing obtained by allowing the blood from a cut artery to spout on the surface of a revolving drum.

Hæmelytrometra.—An accumulation of blood in the cavities of the vagina and uterus. *See* Uterus, Malformations of (*Atresia of Vaginal Orifice*).

Hæmidrosis. *See* Hæmathidrosis.

Hæmin.—The hydrochloride of hæmatin, formed by heating blood with chloride of sodium

and glacial acetic acid and evaporating; its chemical formula is believed to be $C_{32}H_{30}N_4Fe O_3HCl$; its crystals are black in colour and rhombic in shape; and its formation is used as a test for blood stains. See MEDICINE, FORENSIC (*Blood Stains, Hæmin Crystal Test*); PHYSIOLOGY, BLOOD (*Cells of Blood, Chemistry*).

Hæmochromatosis.—A morbid condition in which there is extensive pigmentation of the body, with secondary cirrhosis of the liver and pancreas; there is marked destruction of red blood corpuscles and cachexia; it is probably the same disease as diabetic cirrhosis ("*cirrhose pigmentaire*"). See LIVER, DISEASES OF (*Arteriosclerosis of Hepatic Artery, Hypertrophic Biliary Cirrhosis, Pigmentation of the Liver*).

Hæmochromogen.—Reduced alkaline hæmatin. See PHYSIOLOGY, BLOOD (*Cells of Blood, Chemistry*); PIGMENTS OF THE BODY AND EXCRETA (*Hæmochromogen*).

Hæmochromometer.—An instrument for determining the amount of hæmoglobin in the blood by the tint of the latter.

Hæmocyanin.—The respiratory pigment of the blood of some crustacea and molluscs (*e.g.* the Octopus); it contains copper.

Hæmocytolysis.—A breaking down of the red blood cells.

Hæmocytometer.—An instrument for estimating the number of blood corpuscles per cubic millimetre. See BLOOD (*Enumeration of Corpuscles, Thoma-Zeiss and Oliver Hæmocytometers*); PHYSIOLOGY, BLOOD (*Cells, Red, Enumeration*).

Hæmocytozoon.—A parasitic pigmented protozoon found in the blood corpuscles in malaria. See MALARIA (*Parasitology*); MICRO-ORGANISMS (*Protozoa*).

Hæmodynamometer.—An instrument, such as Oliver's, for determining the blood pressure; a blood-pressure gauge. See PULSE (*Measurement of Arterial Pressure in Man*).

Hæmoferrum.—A preparation of an iron-containing proteid used in anæmia, chlorosis, etc. See CHLOROSIS (*Treatment*).

Hæmogallol.—An iron-containing preparation used in anæmia; it is made from the oxidation of hæmoglobin.

Hæmoglobin. See ANÆMIA, PERNICIOUS (*Symptoms, Percentage of Hæmoglobin*); BLOOD (*Methods of Clinical Examination, Estimation of Hæmoglobin*); CHLOROSIS (*Pathology*); LIVER, PHYSIOLOGY OF (*Elimination of Hæmoglobin*); METEOROLOGY (*High Altitudes, Effect on amount of Hæmoglobin*); PHYSIOLOGY, BLOOD (*Cells, Red, Pigment of*); PIGMENTS OF THE BODY AND EXCRETA (*Hæmoglobin and its Derivatives*); SPECTROSCOPE IN MEDICINE (*Absorption Spectra of Pigments*); URINE, PATHOLOGICAL CHANGES IN (*Proteids, Hæmoglobin*); URINE, PATHOLOGICAL CHANGES IN (*Sediments, Hæmoglobin Casts*).

Hæmoglobinæmia.—Existence of the hæmoglobin of the blood in a state of solution in the serum. See HÆMOGLOBINURIA (*Definition*).

Hæmoglobinometer.—An instrument for estimating the amount of hæmoglobin in the blood; in addition to the hæmoglobinometers already described (*v.* BLOOD, *Estimation of Hæmoglobin*), that invented by Haldane is worthy of mention. In order to obtain an unalterable standard colour Haldane has substituted for the picrocarmine jelly of Gower's instrument a solution of carbonic oxide hæmoglobin, which remains stable for an indefinite time. The technique is the same as in using Gower's instrument, with the exception that the oxyhæmoglobin of the blood examined is converted into carbonic oxide hæmoglobin by allowing coal gas to act upon it. The instrument permits of considerably more accurate readings than the ordinary Gower's hæmoglobinometer.

Hæmoglobinuria.

See also BLACKWATER FEVER; LABOUR, OPERATIONS (*Induction of Premature Labour, Glycerine Injections*); NEW-BORN INFANT (*Winckel's Disease, Hæmoglobinuria Neonatorum*); PARASITES (*Hæmosporidia, Hæmoglobinuria of the Ox*); RABIES (*Symptoms, Urine*); RAYNAUD'S DISEASE (*Complications, Paroxysmal Hæmoglobinuria*); TRANSFUSION (*Dangers*); TROPICS, UNCLASSED FEVERS OF (*Hæmoglobinuric Fever*).

THE term hæmoglobinuria is used to designate the passage of urine containing free oxy- or methæmoglobin in solution, or in the form of granular débris, as distinguished from hæmaturia, the passage of urine mixed with blood. It is the outward manifestation of hæmoglobinæmia, by which is meant the presence of free hæmoglobin in the blood-plasma.

Ponfick has shown that if a solution of hæmoglobin, or laked blood, be injected into the circulation of an animal, until a certain propor-

tion is reached the metabolic processes are able to dispose of the introduced pigment, but that if this proportion be exceeded the excess of hæmoglobin is excreted by the kidneys.

A similar result follows transfusion of the blood of an animal of a different genus, under which circumstances the foreign red corpuscles undergo disintegration, and the blood pigment which they contained appears in the urine. The transfusion of lamb's blood into human patients, which was at one time recommended as a remedial measure, was usually followed by hæmoglobinuria, but this is not the case when transfusion is performed from one human being to another, unless the corpuscles have been previously broken up.

There can be no doubt that hæmolysis is constantly in progress in the body, and that in certain diseases, such as pernicious anæmia, its normal limits are greatly exceeded, and we must suppose that under such conditions the metabolic processes which dispose of the effete blood pigment are not over-taxed, or that the hæmolysis takes place elsewhere than in the general circulation. In the conditions which are attended by hæmoglobinuria, on the other hand, the destructive process goes on with great rapidity, and apparently in the general circulation.

Jaundice of greater or less degree frequently accompanies hæmoglobinuria, and it may be looked upon as another manifestation of hæmoglobinæmia. This fact was held to supply one of the strongest arguments for the existence of a hæmatogenous variety of jaundice, due to the change of blood pigment into bile pigment without the intervention of the liver. More recent researches, however, tend to show that even in these cases the jaundice is obstructive, and results from changes in the consistence of the bile, partly due to the formation of an excess of bile pigment, and partly to an increased secretion of mucus, as a result of irritation of the biliary passages.

Toxic Hæmoglobinuria.—A great variety of toxic substances possess the hæmolytic power requisite for the production of hæmoglobinuria. And of these the most important, because it is so extensively used as a drug, is potassium chlorate, which appears to share with other chlorates the additional property of converting oxy- into methæmoglobin in the circulation, and even within the red corpuscles. Arseniuretted hydrogen is also a hæmolytic poison, and Eitner and others have recorded cases in which hæmoglobinuria has resulted from the inhalation of hydrogen containing this gas as an impurity. Amongst other such poisons may be mentioned toluylene-diamine, the effects of which upon animals have been carefully studied by Stadelmann, William Hunter, and others; nitrobenzol, hydrochloric and sulphuric acids, carbolic acid, pyrogallol, naphthol, and sulphuretted hydrogen.

Even quinine must be mentioned among the substances which are credited with giving rise to hæmoglobinuria, especially in malarial cases (*see* article "Blackwater Fever" in vol. i. p. 376).

Bostroem has described some cases of poisoning by a mushroom (*Morchella esculenta*) in which hæmoglobinuria was a prominent symptom.

Hæmoglobinuria in Fevers.—Hæmoglobinuria may also occur in the course of various specific fevers and in septic diseases. It is seldom observed, and as a rule in severe cases. Thus Immermann met with it in a relapse of typhoid fever, and Cnopf and Huebner have described its occurrence in scarlatina. Hayem and Robin have seen hæmoglobinuria as a complication of acute rheumatism, and the present writer has seen a very temporary hæmoglobinuria in a case of this nature, and also in one of lobar pneumonia. In both instances it was of very short duration; the urine was pink and quite limpid, containing hæmoglobin, but no blood corpuscles. In malarial fevers hæmoglobinuria sometimes occurs, and it is a prominent symptom of the so-called "blackwater fever," which owes its name to this circumstance.

Hæmoglobinuria in the Lower Animals.—Hæmoglobinuria is a prominent symptom of an epidemic disease which occurs in cattle, and in which Babes found a micrococcus-like organism in the blood, and especially in the kidneys.

Horses also are liable to a peculiar affection which attacks heavy draught animals, on resuming work after a few days' rest and good feeding. Its onset appears to be favoured by cold. Hæmoglobinuria is a prominent symptom, and it is attended by an acute form of myositis. The attacks may be prevented by reducing the food and giving the animals some exercise during the resting period.

In this connection it is interesting to note that Chvostek found that hæmolysis was more easily produced in horses than in other animals.

Infantile Hæmoglobinuria.—Winkel has described a remarkable epidemic which occurred among new-born infants in the lying-in institution in Dresden, in which hæmoglobinuria was a prominent symptom. In all, twenty-three infants were attacked in quick succession, and of these no less than nineteen died. The onset was usually on or about the fourth day of life, and the average duration of the illness was thirty-two hours. The other leading symptoms observed were cyanosis, some degree of jaundice, which was often very slight, coldness of the extremities, and unconsciousness. There was no febrile disturbance in any case, but rather a rapid fall of temperature.

The blood was found to have a treacly consistence and a dark brown colour. There was a marked leucocytosis, and much granular detritus was present.

The post-mortem appearances recalled those met with in cases of potassium chlorate poisoning, and included deep pigmentation of the renal cortex, hyperæmia of the pancreas, enlargement of the spleen and mesenteric glands, dilatation of the stomach, and ecchymoses in the stomach and intestines, with swelling of Peyer's patches.

Numerous micrococci were found in the blood, kidneys, and intestine.

Winkel considered that umbilical infection might be excluded—no medicinal or dietary cause could be traced, and it is noteworthy that all but five of the affected infants were breast-fed, and the mothers showed no impairment of health. Sporadic cases, apparently of the same nature, have since been recorded by Sandner and Baginsky.

Paroxysmal Hæmoglobinuria.—Of the conditions which give rise to the symptom under discussion, the most remarkable, and in many respects the most interesting, is the affection known as paroxysmal hæmoglobinuria, which appears to be a disease *sui generis*.

The earliest clear description of this malady, as one characterised by the intermittent passage of urine deeply coloured by blood pigment and containing hæmoglobin débris, but free from red blood corpuscles, is contained in a paper by George Harley in the *Medico-Chirurgical Transactions* for 1865, which is immediately followed by a record of some further cases by W. H. Dickinson. It is true that the disease does not appear to have wholly escaped the notice of earlier observers, and reference may be made to the description of a case of intermittent albuminuria and chromaturia by Dressler, published in 1854. In a paper by Alexander Marcet, published in 1823, reference is made to a case, apparently of this nature, in which the vaso-motor phenomena were so well marked that it may be regarded as an example of Raynaud's disease.

Paroxysmal hæmoglobinuria is a decidedly rare disease. It commences usually in adult life, before middle age, but it is also met with in children. A very great majority of all the sufferers are males, the recorded cases in females being very few in number.

Malaria and syphilis have been regarded as important predisposing causes, but the more recent investigations suggest that malarial antecedents play a less important part than the earlier observers supposed; whereas it has been shown that a history of syphilis is to be obtained in a large proportion of cases, and children who suffer from the disease have usually suffered from congenital syphilis also.

The initial attack is not infrequently ascribed to some unusually severe exposure, and beyond question cold is by far the most potent exciting cause of the individual paroxysms. In some cases the effect of cold appears to be aggravated by exertion, and there is a remarkable group of cases in which muscular exertion of a particular kind appears to be the sole provoking cause of attacks. Since, however, it is doubtful whether these cases are rightly included in the present category, it will be best to discuss them separately.

The influence of cold is best shown by the fact that in susceptible subjects an attack may be started by a cold bath, and even by the immersion of portions of the body, such as the hands or feet, in ice-cold water, for a longer or shorter period. However, individual patients differ remarkably in their susceptibility to such influences, and a degree and duration of exposure which will in one case inevitably provoke an attack, will in another have no such result.

In addition to hæmoglobinuria a variety of more general symptoms accompany the attack, such as pains in various parts of the trunk and limbs, rigors, febrile disturbance, and, in the later stages, sweating. Headache is often present and may be severe, and gaping or yawning is a common early symptom. Nausea, anorexia, vomiting, and a peculiar sensation of difficulty of swallowing, are also described or observed; and in some cases an urticarial rash appears during the attack.

Among the most conspicuous phenomena are vaso-motor disturbances, such as coldness of the extremities, and "deadness" of the fingers and toes, which are the seat of tingling and numbness, and appear white and bloodless, or even assume a purple tint. These changes are attended by a rise of blood pressure due to contraction of the peripheral vessels. In a word, a condition is developed which is indistinguishable from the slighter forms of Raynaud's disease, and even the more severe Raynaud phenomena are sometimes met with in association with hæmoglobinuria. It would, perhaps, be more correct to say that hæmoglobinuria is sometimes seen in Raynaud's disease, for whereas vaso-motor disturbances of minor degree are usually observed in paroxysmal hæmoglobinuria, it is only in a few cases of typical Raynaud's disease that hæmoglobinuria occurs.

However, the association is sufficiently striking to warrant the supposition that there is some intimate relationship between the two conditions, rather than that, as Bristowe and Copeman suggested, their association is merely due to the fact that exposure to cold is the common exciting cause of both maladies. Both diseases are decidedly rare, and the probability of their accidental association in the same individual is consequently small.

Recent observations suggest that the vaso-motor disturbances play an important part in the causation of paroxysmal hæmoglobinuria, a second factor being an abnormal condition of the blood, and it seems possible that in cases of

Raynaud's disease without hæmoglobinuria this second factor is wanting.

Tenderness in the region of the liver and spleen have occasionally been observed, and even palpable enlargement of those organs.

The general symptoms above mentioned vary in individual cases, and only a selection of them is usually present. However, in any given case the symptoms tend to be fairly constant, each successive paroxysm resembling those which have gone before. Thus one patient stated that he always experienced numbness of all his fingers and of his great toes, whilst in another case the paræsthesia was always most marked in the ring and little fingers, and especially in those of the left hand. Again, the distribution of the pains in the trunk and limbs tends to repeat itself. In one case the attack will always be ushered in by a pain down the back, in another by aching in the loins and thighs.

The number and intensity of the symptoms present are also apt to vary with the severity of the attack, which, again, is usually in proportion to the degree and duration of the antecedent exposure to cold.

Unless some permanent renal affection be present the urine shows nothing abnormal in the intervals between the attacks. The onset of hæmoglobinuria usually occurs an hour or so after the exposure, and it may be preceded by a simple albuminuria. In some instances the urine has contained an excess of urobilin after the attack, but I have never observed this. Possibly it only occurs when the attack is accompanied by jaundice.

The characteristic urine is usually acid and often strongly so, but in some recorded cases it has been constantly alkaline. The specific gravity is as a rule lowered (1010-1015).

Its colour varies from ruby red to that of porter, and in thick layers it may appear almost black. The blood pigment may be present in the form of oxy- or methæmoglobin, and most frequently the spectroscope shows the bands of both those pigments.

The presence of methæmoglobin is evidenced by its characteristic band in the red, and that of oxyhæmoglobin by the intensity of its bands near D and E, which far exceeds that of the corresponding bands of the spectrum of methæmoglobin.

Copeman found that, in his case, when the urine was drawn off by catheter at short intervals it showed the bands of oxyhæmoglobin only, whereas when it remained longer in the bladder it contained methæmoglobin. In some cases examined by the present writer, the band in red of methæmoglobin was clearly visible immediately after the urine was passed, even when the bladder had been emptied not long previously, but the specimens were not drawn off by the catheter. Copeman further believes that, on standing, a portion of the excreted hæmoglobin undergoes a further change to acid hæmatin, and this is one of the many points connected with this disease which calls for further investigation. The presence of methæmoglobin is no special feature of the urine of paroxysmal hæmoglobinuria, nor, indeed, of hæmoglobinuria in general as distinguished from hæmaturia. Hoppe Seyler stated that blood pigment is always passed in the urine in the form of methæmoglobin, and that it changes to oxyhæmoglobin on standing. That such a change takes place in ordinary smoky urines it is easy to convince one's self, but the rate of change varies widely in different specimens.

The most striking microscopical feature of the urine is the absence of red blood corpuscles. It is true that a few are sometimes seen in the sediment, but they bear no proportion to the colour, and their presence is not improbably due to temporary changes in the kidney resulting from the unwonted calls upon them. Such renal changes have been observed in animals after transfusion of foreign blood.

The urine deposits a copious dark brown sediment, which consists of granular hæmoglobin débris, and sometimes of urates mixed with such débris. Casts of renal tubules composed of brown granules are usually present in large numbers, as they also are in the urine of the various forms of toxic hæmoglobinuria and of transfusion cases.

Crystals of calcium oxalate are very frequently seen in such urines, but as yet there is no evidence that their presence has any special pathological significance.

The free passage, through kidneys ordinarily proteid-tight, of such large quantities of proteid material is a matter of considerable interest, which may be compared with the excretion in the urine of other foreign proteids, such as egg albumin, after injection into the circulation of an animal. Some recent researches of Brodie suggest, however, that the physical properties of hæmoglobin may aid in its elimination, as it is found to pass more readily through a perfused kidney or a Martin's filter than either serum albumin or globulin.

Serum albumin is also usually present in small amount in the dark urine of the paroxysm, and some albuminuria may persist for a time after the hæmoglobinuria has passed away. Again, in some cases slighter exposures are followed by albuminuria, and more severe ones by hæmoglobinuria; and Ralfe suggested that the transitory or paroxysmal albuminuria of apparently healthy individuals is really a minor manifestation of the disease under discussion—its "petit mal," as Bristowe happily styled it.

The low specific gravity of the urine passed during the paroxysm suggests a diminished excretion of the heavier urinary constituents, due to blocking of the renal tubules with hæmoglobin débris, and in some forms of toxic

hæmoglobinuria the renal secretion has been completely suppressed. Boas was inclined to attribute many of the attendant symptoms to this cause, and the effect of the attack upon nitrogenous excretion is a point which calls for investigation.

Blood counts made before and during the attacks show a marked fall in the numbers of the red corpuscles. Copeman has observed falls varying from 129,000 to 824,000 per cubic millimetre. The destruction is most active at the very commencement of the attack, before the hæmoglobin appears in the urine.

Hayem observed, and Chvostek has confirmed the observation, that blood drawn during the paroxysms clots with unusual rapidity, and that the clot quickly liquefies again. This peculiarity is not met with in blood taken during the free intervals.

The presence of free hæmoglobin in solution in the serum during the attack has been repeatedly observed, both in serum from which blood-clot has separated and in that obtained from blisters. Copeman has occasionally observed the formation of crystals of hæmoglobin in serum which has been allowed to stand.

One of the most obvious microscopic changes is the absence of rouleaux formation by the red corpuscles. Poikilocytosis is sometimes noticed, and particles of free pigment are seen in the plasma. The blood platelets are usually increased in number. As a rule there is no leucocytosis.

Ehrlich, Boas, and other investigators have laid special stress upon the presence of decolorised red corpuscles (*Blutshatten*) as a characteristic feature.

During the exposure which excites the attack there is, as Bristowe and Copeman pointed out, an initial fall of temperature, which is quickly followed by a decided rise, during which 101° or even 103° F. may be reached. Shortly after the maximum has been reached a rapid fall commences, warmth returns to the extremities, and the urine regains its normal character, although slight albuminuria may outlast the hæmoglobinuria for a time. The paroxysms commonly last some five or six hours, but their duration is to some extent proportional to their severity. Occasionally febrile disturbance is absent, and the temperature may even remain subnormal throughout.

The patient is left somewhat anæmic, especially if several attacks have followed each other in quick succession, but the destroyed blood corpuscles are quickly replaced. As in other varieties of hæmoglobinuria, some degree of jaundice may follow the attack, but this is usually slight in character, and is by no means constantly present.

As might be expected, the attacks are most frequent in winter, and many patients enjoy complete immunity from them during the summer months. Even in winter the disease remains in abeyance as long as the patient is protected from cold, as, for example, during a stay in hospital.

Seeing that in the intervals between the paroxysms the patient enjoys practically unimpaired health, except during the short period required for the replacement of the destroyed blood, and since the attacks themselves are not attended by danger, it is not remarkable that the disease has practically no fatal tendency, and may continue for many years without serious detriment to health.

Pathology.—It will be seen from what has gone before that the main features of the attack of paroxysmal hæmoglobinuria are—1st, the active hæmolysis which occurs on exposure to cold; and, 2nd, the vaso-motor phenomena by which it is accompanied.

It is probable that many of the minor symptoms are direct results of hæmoglobinæmia. If one refers back to the records of cases of transfusion of lamb's blood into human beings, one is struck by the similarity of the symptoms observed with those of the disease under consideration. Rigors and febrile disturbance were prominent effects, and among other symptoms mentioned, in addition to hæmoglobinuria, were pains in the back and lumbar regions, vomiting, dyspnœa, sweating, and urticarial eruptions. This urticaria followed quickly upon the transfusion, and therein differed from that which is so common a result of the modern serum treatments. It is further of interest to note that rigors, fever, and sweating were observed by Eitner in his cases of poisoning by arseniuretted hydrogen.

On the other hand, rigors and febrile disturbance may follow transfusion from man to man, and were also observed in some cases in which transfusion of milk was resorted to.

One of the main points to which investigation has been directed is the obvious fact, that in the subjects of paroxysmal hæmoglobinuria the red corpuscles show an abnormal tendency to undergo rapid destruction under influences which in ordinary individuals are insufficient to bring about such a result. This susceptibility of the red corpuscles is not a congenital condition, but is acquired at some period of the patient's life, often apparently as a result of an exceptional exposure, and when once acquired it tends to persist.

The researches of Ehrlich, Chvostek, and others, have clearly demonstrated that in the sufferers from paroxysmal hæmoglobinuria the corpuscles are not unduly liable to disintegration, outside the body, under the influence of cold, and Ehrlich was consequently inclined to attribute their intravascular disintegration, under the influence of exposure, to the secretion of a hæmolytic ferment by the walls of the vessels in the part cooled.

Chvostek has shown, on the other hand, and

Mannaberg and Donath have recently confirmed his observation, that the red corpuscles of these patients are abnormally sensitive to mechanical influences, such as shaking the blood in a tube, or the jarring of a centrifuge which does not run smoothly. Mannaberg and Donath have further shown that they are broken up with unusual readiness under the influence of carbon dioxide.

Chvostek also found that in repeating the classical experiment of Ehrlich, viz. placing an elastic ligature upon the patient's finger and immersing it in ice-cold water, the cooling was not necessary for the production of local hæmoglobinæmia, but that the simple arrest of the circulation leads to this result. Following Murri, he looks upon the vascular spasm as playing an equally important part with the abnormal blood condition, the presence of both factors being necessary for the production of the attack. Such a theory will explain very well the relationship of Raynaud's disease to paroxysmal hæmoglobinuria, the cases in which the former is present without hæmoglobinuria being those in which the second factor, viz. the undue liability of hæmolysis, is absent. In support of his view that the vaso-motor disturbances, aided by the resultant presence of carbon dioxide in excess, brings about the destruction of the abnormally frail blood corpuscles, Chvostek adduces the fact that he was able to cut short the attacks by the administration of amyl nitrite at the earliest stage. Moreover, he found that in horses, whose blood corpuscles appear to be less stable than those of other animals, he was able to bring about a degree of hæmoglobinæmia by the induction of vascular spasm by electrical stimulation of the upper portion of the cervical cord.

Against this must be set the failure of Mannaberg and Donath to cut short the attacks by amyl nitrite in their case, and these authors object to Chvostek's theory that it is not at all evident in what way vascular spasm can place the blood under conditions at all comparable to those present in the shaking experiments, nor do they think that the facts can be more satisfactorily explained by a theory based upon their results with carbon dioxide, venosity of the blood being held wholly responsible for the hæmolysis.

The experiment with the ligature of a finger shows that local hæmolysis may take place in such cases in the peripheral circulation, and the seat of the blood destruction during the paroxysm has been variously located, several observers regarding the renal circulation as the most likely locality. Against this latter view is the presence of free hæmoglobin in the serum generally, in spite of its abundant excretion in the urine. Chvostek's observations led him to the opinion that only a small proportion of the red corpuscles are unduly subject to disintegration under mechanical influences, and if this be so, it points to the tendency to break up being inherent in the corpuscles themselves rather than dependent upon any abnormality of the medium in which they are suspended.

Seeing that so many poisons possess hæmolytic powers, and are capable of causing hæmoglobinuria, the possibility suggests itself that such a toxic substance may be present in the blood of these patients in amounts too small for the direct manifestation of its hæmolytic action under ordinary conditions, but which, nevertheless, is able to bring about blood destruction when aided by the other conditions which excite an attack.

However, such an hypothesis receives no countenance from some remarkable observations of Mannaberg and Donath, who found that whereas the serum of blood obtained from a patient during an interval, and immediately centrifugalised, caused no hæmolysis when added to healthy blood, the serum of an attack, which was slightly coloured by hæmoglobin, became more deeply coloured, and showed the absorption bands more clearly, after it had been added to healthy blood and again separated. A similar hæmolytic action was exercised by serum in which hæmolysis had been mechanically brought about.

The fact that hæmoglobinuria is an occasional symptom of malarial fever, and the part which ague appears to play as a predisposing cause in some cases of paroxysmal hæmoglobinuria, suggest a community of origin, and there are features in the attacks which lend colour to this hypothesis; but, on the other hand, the paroxysms of hæmoglobinuria are in no sense periodic, and their recurrence is strictly dependent upon exposure to the exciting cause. The more recent observers are disposed to agree with Murri in regarding syphilis as a far more important predisposing cause, and a history of malaria is comparatively seldom to be obtained in cases met with in this country.

The scanty number of recorded post-mortem examinations of the subjects of paroxysmal hæmoglobinuria throws practically no fresh light upon the problem of its pathology, the changes met with being similar to those seen in other conditions accompanied by hæmolysis and hæmoglobinuria. In some cases interstitial changes have been observed in the kidneys, which possibly have been results of the unusual work which these organs are at intervals called upon to perform. That this is not entirely without effect upon the kidneys is suggested by the presence of small quantities of serum albumin, and occasionally of a few red corpuscles in the urine during the attacks.

It will be evident, then, that in spite of the large amount of careful investigation which has been directed upon its study the pathology of paroxysmal hæmoglobinuria is still to a large

extent obscure, and that no theory of its causation which has as yet been put forward affords a full and satisfactory explanation of the observed phenomena.

Treatment.—Removal of the patient from the conditions which provoke the attacks is one of the prime desiderata in the treatment of the disease, and, when circumstances permit, residence in a climate in which no extremes are encountered, or at least a winter sojourn in a warm climate, is clearly indicated, and may serve to procure complete immunity. In the majority of instances measures directed to the cure of the disease, and to enabling the patient to encounter exposure to cold with impunity, have less satisfactory results. A complete cure has occasionally been brought about by antisyphilitic treatment in the hands of Murri and others, and good effects have sometimes followed the administration of quinine in full doses.

In the treatment of the individual paroxysms warmth is indicated, and a warm bed and warm drinks add greatly to the comfort of the patient, even if they do not curtail the attack. The administration of nitrite of amyl, which Chvostek found effectual in cutting short the paroxysm, should receive a trial, but as the hæmolysis occurs at so early a stage, it is obvious that, if it is to produce any effect, it must be given at the very first onset of the symptoms. Nitroglycerine might also prove of service as a prophylactic. Copeman recommends the avoidance of alcoholic drinks on account of the temporary dilatation of the superficial capillaries which they produce, which may bring about a slight lowering of the body temperature.

Hœmoglobinuria from Exertion.—The cases in which hæmoglobinuria is induced by muscular exertion are few in number, but of great interest. As they differ in certain not unimportant respects from the ordinary cases of paroxysmal hæmoglobinuria, they are best considered provisionally as constituting a separate group. Examples have been recorded by Fleischer, Kast, Robin, Köster, Lee Dickinson, and others. In some instances at any rate the attacks are frequently repeated, and may be set up even by a very moderate exertion, in others a single attack follows a severe muscular strain, such as is involved in running a three-mile race.

It has been demonstrated in some cases that such exposure to cold as might be expected to induce a paroxysm in an ordinary case of paroxysmal hæmoglobinuria was not capable of producing such an effect, even when an attack followed a moderate walk.

Perhaps the most curious feature of these cases is the apparent powerlessness of other forms of exertion than those involving leg-work to induce the attacks. Marching, walking, running, and in one of Lee Dickinson's cases a hard game of lawn tennis, are the recorded causes, and a patient who develops hæmoglobinuria after a walk may be able to exert himself at wood-chopping with impunity. Another noteworthy feature is that the attendant symptoms which are usually so prominent in paroxysmal hæmoglobinuria are usually absent, and the abnormal coloration of the urine may alone attract attention. Köster's patient, however, complained of coldness of the extremities, chills, and slight headache, but showed no febrile disturbance. This observation is opposed to the view which attributes many of the attendant symptoms to hæmoglobinæmia, but the difference may be merely one of degree, for in some of the exertion cases the amount of hæmolysis, as evidenced by the condition of the urine, seems to have been comparatively slight. In a case described by Bastianelli albuminuria without hæmoglobinuria was observed after exertion on some occasions. Köster's patient, who had acquired syphilis four years previously, recovered completely under antisyphilitic treatment, and in Robin's case recovery followed treatment by rest, tonics, and a restricted diet. In this latter instance, before recovery was complete, exertion which would formerly have induced hæmoglobinuria caused only slight albuminuria.

Hæmoglobinuric Fever. *See* Blackwater Fever ; Tropics, Unclassed Fevers of (*Fevers of Mixed Origin*).

Hæmohydronephrosis.—A cystic condition of the kidney, the contents of the dilated cavities being blood and urine.

Hæmokonia.—This term (Gr. αἷμα, blood, and κονία, dust) has been applied to small refractive bodies found in the blood and ascribed to the breaking down of the corpuscles; blood-dust.

Hæmol.—A deoxidation product of hæmoglobin, used in anæmia ; hæmogallol. *See* Chlorosis (*Treatment, Special*).

Hæmolymph Glands.—Organs intermediate between lymphatic glands and the spleen ; they consist of lymphatic glands with large blood sinuses round them ; and they are found in certain animals. They produce lymphocytes, store up iron, and dispose of disintegrating corpuscles. *See* Physiology, Blood (*Source of the Blood Constituents*).

Hæmolysins.—Blood-dissolving principles. *See* Hæmolysis ; Immunity.

Hæmolysis.—The process of breaking down old red blood corpuscles and eliminating their pigment, which goes on in the liver, and possibly also in the spleen and other organs and tissues. *See* Physiology, Blood (*Fate of the Blood Constituents*) ; *also* Hæmoglobinuria (*Definition*) ; Immunity.

Hæmomanometer.—An instrument for the determination of blood pressure.

Hæmomenas Præcox.—The parasite of malignant or æstivo-autumnal malarial fever. *See* MALARIA (*Blood in Malignant Fever*).

Hæmometer. *See* HÆMOGLOBINOMETER.

Hæmo-Pericardium.—The presence of effused blood in the pericardial sac. *See* AORTA, ANEURYSM (*Course and Terminations, Rupture*); CHEST, INJURIES OF (*Wounds of Heart and Pericardium*); HEART, MYOCARDIUM AND ENDOCARDIUM (*General Pathology, Morbid Processes, Hæmorrhage*).

Hæmo-Peritoneum.—The presence of effused blood in the peritoneal cavity.

Hæmophagocytes. — The white corpuscles in the blood as distinguished from those in the tissues.

Hæmophilia.

See also ADRENAL GLANDS (*Adrenalin*); ASPIRATOR, USES OF (*Abscesses in Hæmophilic Patients*); CAUTERY (*Red Heat, in Hæmophilia*); HÆMATEMESIS (*Causes*); HEREDITY (*Inheritance of Disease*); JOINTS, DISEASES OF (*Bleeders' Joint*); LEUCOCYTOSIS (*Leucopenia*).

HÆMOPHILIA, or the congenital hæmorrhagic diathesis, is a congenital hereditary disease, characterised by a liability to spontaneous hæmorrhages, to excessive bleedings from trivial causes, and to joint affections. The morbid tendency persists throughout the life of the affected individual, who is termed a "bleeder."

Etiology.—Heredity and sex are important factors in the production of the disease, yet its actual cause is unknown. Thus the disease occurs in males, females being very rarely affected. Transmission does not as a rule take place directly from father to son, but almost always through seemingly healthy members of the female line, who are often exceedingly fertile, to the male descendants. A somewhat similar mode of transmission of disease is observed in pseudo-hypertrophic paralysis, Friedreich's ataxia, and occasionally in diabetes insipidus and colour blindness. Apart from heredity, there is no obvious cause for hæmophilia, which is met with in all the human races, in various countries and in all ranks of society. Consanguinity of the parents, and a history of gout in the ancestors, are probably merely accidental circumstances.

Morbid Anatomy. — Post-mortem there is anæmia of the tissues and organs, and in many instances also ecchymoses, blood extravasations into the organs, or effusions into the body cavities. Other changes, such as fatty degeneration of organs, thinness of the arterial coats, and abnormally narrow vessels, are not constant, nor are they causal factors. The joints may present morbid appearances from hæmorrhages into their cavities, but in more chronic cases there is thickening of the synovial membrane, with fibrillation, and degeneration of the articular cartilage, the changes in short resembling those seen in rheumatoid arthritis. There is no proof that the blood differs from the normal as regards its chemical constitution. When a bleeding first starts the microscopic appearance of the blood is quite normal, and coagulation is normal also. But if the blood be examined after a severe hæmorrhage, there will necessarily be signs of anæmia, and the blood "resembles water in which fresh meat has been washed and scarcely stains linen" (Legg). Coagulation, too, is then very deficient, or does not occur at all. The essential nature of the disease, in short, is unknown, though generally held to consist either in some abnormality of the blood-vessels, or in an abnormal state of the blood whereby clotting is retarded or altogether in abeyance.

Symptoms are classified under three headings: (1) Spontaneous or traumatic external hæmorrhages; (2) Interstitial hæmorrhages; (3) Joint affections.

1. In a small percentage of cases the disease manifests itself soon after birth by severe umbilical hæmorrhage. As a rule, however, the child is about a year old before any signs appear, the subjects of hæmophilia being usually quite normal as regards their general appearance, physiognomy, nutrition, etc. The earliest sign is often a severe external hæmorrhage after some trivial wound, and as the boy grows older he suffers frequently from these uncontrollable bleedings after various forms of traumatism, such as slight cuts, tooth extraction, circumcision, opening of abscesses, etc. The hæmorrhage is usually capillary, and is characterised by its severity and the great difficulty with which it is checked, blood oozing away slowly but steadily, it may be for days or weeks in succession. Or the bleeding may be spontaneous, and is then more often from the mucous and serous surfaces. In early life these bleedings often occur as severe epistaxes, less commonly as bleedings from the gums, and after the beginning of the second decade there is a greater tendency to intestinal, urethral, renal, gastric, or pulmonary hæmorrhages, than in infancy. Effusion of blood into the peritoneal cavity is occasionally seen.

2. The interstitial hæmorrhages are either spontaneous or occur after traumatism. In the

former case there are petechiæ or ecchymoses in the skin during infancy or later life, and hæmorrhages in the mucous membranes, tissues, and organs. The ecchymoses in no way differ in appearance from similar conditions found in persons with an acquired hæmorrhagic diathesis. Traumatic ecchymoses are common, and allied to them are hæmatomata, which may attain the size of a man's head.

3. Joint affections do not usually arise till the fourth or fifth year, and the larger joints are the more commonly affected. Spontaneous or traumatic hæmorrhage occurs into the joint cavity (hæmarthrosis), and there is an inflammatory infiltration of the periarticular tissues. Thus the joint appears swollen, is painful, especially on movement, and the overlying skin is often somewhat discoloured. The onset of the swelling is often sudden, and accompanied by a rise of the temperature to 103°-104° F. The swelling usually disappears in the course of a few weeks, but tends to recur. In consequence of repeated intra-articular hæmorrhage changes occur in the joint structures, giving rise to appearances closely resembling rheumatoid arthritis, and in later stages the ends of the bones are much thickened, and the joint is more or less anchylosed, flexed, and useless.

Diagnosis is easy in well-marked cases. The milder varieties may present more difficulty; important points to remember are that the disease is hereditary and mainly affects males, that the first signs appear as a rule before the tenth year, and that the disease lasts throughout life. It may be mistaken for—(i.) *The hæmorrhagic diseases of the new-born.* Idiopathic umbilical hæmorrhage affects both sexes equally, and leaves no subsequent liability to excessive hæmorrhages. Melæna neonatorum usually occurs within a few days of birth, is due to gastric or intestinal ulceration, and is often fatal within a week. Some forms of congenital syphilis and the hæmoglobinuria of Winkel's disease are readily differentiated, the latter being accompanied by cyanosis, jaundice, and somnolence, and being probably an infective disease. (ii.) *Scurvy,* the various forms of *purpura* and the hæmorrhages of gout, leucocythemia, etc., are as a rule easily distinguished. (iii.) Lastly, the joint affections must not be confounded with *rheumatism, gout, rheumatoid arthritis,* or *tubercular disease* of the joints.

Prognosis. — The younger the patient the graver is the prognosis; but with care adult life is often attained, and the hæmorrhagic tendency possibly diminishes after middle life.

Treatment. — If there is known to be an hereditary tendency to the disease, the children, and especially the boys, should be carefully reared, and all operative measures avoided, with the exception of vaccination, which is hardly ever attended with special risk. Older children and adults have usually been warned by their relatives of their morbid tendency, but some bleeders are peculiarly reticent in admitting that they are sufferers; hence, if hæmophilia be suspected, mere denial thereof should not entirely allay our suspicions. If traumatic hæmorrhage has occurred, general surgical measures must be employed to arrest the bleeding; operative interference, however, causes hæmorrhage as uncontrollable as that from the initial lesion. Compression, combined, if necessary, with plugging of the wound, is the most efficient treatment, especially if complete rest be observed at the same time. Styptics are useless, but freezing with ice or ethyl chloride has sometimes acted well. Internal medicaments, *e.g.* chloride of calcium, ergot, or acetate of lead, may also be tried, but are of more use for checking bleedings from mucous surfaces. Substances, such as nucleic acid, yeast, thymus, and other preparations containing nuclein, are recommended on account of the fact that they produce leucocytosis. Transfusion may be resorted to if other measures fail. Treatment which appears beneficial in one case is often useless in another, in which the bleeding continues unchecked, only ceasing when syncope ensues, or even causing the death of the patient. Joint swellings are best treated by means of compression and immobilisation. After a bleeding the patient usually remains somewhat anæmic for some months, and the administration of iron, cod-liver oil, and other tonics, with attention to the hygienic surroundings, is advisable.

[A case has been reported by Ballantyne (*Journ. Amer. Med. Assoc.* xxxvii. 503, 1901), in which chloride of calcium was given during pregnancy to a woman who had given birth to two hæmophilic male infants; the pregnancy during which the calcium was given ended in the birth of a perfectly healthy male child; post-partum hæmorrhage, which had been present in the former labours, was absent in this.]

Hæmoporphyrinuria. *See* DERMATITIS HERPETIFORMIS (*Morbid Anatomy and Pathology*); HÆMATOPORPHYRINURIA.

Hæmoproteus Danilewskii. *See* PARASITES (*Hæmosporidia*).

Hæmoprotozoa. *See* MALARIA (*Parasitology*).

Hæmoptysis.

See also AORTA, THORACIC, ANEURYSM (*Course and Terminations, Rupture*); BRONCHI, BRONCHIAL GLANDS (*Enlarged Glands, Symptoms*);

Bronchi, Bronchiectasis (*Chronic, Clinical Phenomena*); Chest, Injuries of (*Viscera, Lungs, Hæmoptysis*); Children, Clinical Examination of (*Respiratory System, Sputum*); Gout (*Irregular, Respiratory System, Hæmoptysis*); Hæmatemesis (*Diagnosis*); Heart, Myocardium and Endocardium (*Symptomatology, Pulmonary Conditions, Embolism*); Hysteria (*Respiratory Disorders*); Lung, Tuberculosis of (*Clinical Features, Lung Symptoms, Hæmoptysis*); Lungs, Gangrene of (*Symptoms, Hæmoptysis*); Lungs, Vascular Disorders (*Pulmonary Embolism, Clinical Features*); Lungs, Parasitic Affections of (*Aspergillosis*); Pneumonia, Clinical (*Clinical Features, Sputum*); Typhoid Fever (*Complications and Sequelæ*).

Definition.—The expectoration of blood from the lungs or lower air-passages (larynx, trachea, and bronchi).

Pathogenesis.—The older writers were in the habit of using the terms *Bronchorrhagia* and *Pneumorrhagia* as signifying in the one case effusion from the bronchial mucous membrane, and in the other escape of blood from the pulmonary vessels proper. Laennec was of opinion that slight hæmoptyses were bronchial in origin, while all grave hæmorrhages were due to pulmonary apoplexy, the result of vascular tension in the lungs themselves. In this he was in error. Pulmonary apoplexy is due to embolism, and is a comparatively rare cause of hæmoptysis.

All pathological evidence goes to prove that in the vast majority of cases of hæmoptysis—both slight and severe—the pulmonary and not the bronchial vessels are to be regarded as the source of the hæmorrhage. This is only what we would theoretically expect when we consider how badly supported the pulmonary vessels are as compared with those of the bronchial mucous membrane. The hæmorrhage itself is due either to escape of blood by *diapedesis*, the result of active or passive hyperæmia of the pulmonary, and to a much less extent of the bronchial capillaries; or, on the other hand, to *rupture of a blood-vessel*, generally a branch of the pulmonary artery, the result of some pathological condition (*e.g.* ulceration or aneurysm) of the vascular wall.

Etiology.—A. *Diseases of the Lungs.—Pulmonary Tuberculosis.*—This, especially in its more chronic forms, is *by far the most important cause of pulmonary hæmorrhage.*

Sir Thomas Watson says: "If a person spits up blood who has received no injury to the chest, in whom the uterine functions are healthy, and who has no disease of the heart, the odds that there are tubercles in the lungs of that person are fearfully high."

Hæmoptysis occurs in about 60 per cent of all cases of pulmonary tuberculosis; the Inpatient Brompton Report gives 62 per cent; Dr. Williams (1000 private cases) gives 57 per cent; Dr. Wilson Fox, 54 per cent. Of 200 successive cases admitted by the writer into Brompton Hospital, hæmoptysis occurred in 70 per cent. It is extremely rare as a symptom of acute miliary tuberculosis. Hæmoptysis occurring in pulmonary tuberculosis is due to one of the following causes:—

(1) *Hyperæmia and Rupture of Capillaries.*—Occurs mainly in the early stages of tuberculosis. Such hæmorrhages are usually very slight, and are indicative of congestive and inflammatory states of the pulmonary tissue. Dr. Kingston Fowler regards capillary hæmorrhage as probably the cause of the streaks and small blood-clots which appear from time to time in the sputum in the course of chronic pulmonary tuberculosis.

(2) *Tubercular Infiltration with subsequent Ulceration and Perforation of a Branch of the Pulmonary Artery.*—Tubercle bacilli have been demonstrated by Koch, Weigert, and Percy Kidd in the thickened and infiltrated vessel walls in connection with commencing caseation. The ulcerative process occasionally leads to perforation of an artery of some magnitude, and in this way may induce very profuse hæmorrhage. This is probably the cause of the severe hæmoptyses which occasionally occur in the earlier stages of pulmonary tuberculosis, particularly when associated with rapid caseation. Fortunately such a result is comparatively rare, owing to the fact that closure of the vessel by thrombosis and endarteritis obliterans generally takes place. In the more rapid forms of tuberculosis, the vascular wall becomes softened and infiltrated before the thrombotic process is complete, and rupture takes place, the vessel being no longer able to withstand the force of the blood pressure within.

(3) *Rupture of an Aneurysm of a Branch of the Pulmonary Artery.*—This is by far the most common cause of profuse hæmoptysis, and occurs most frequently in the stage of excavation. In 30 out of 35 consecutive fatal cases occurring at Brompton Hospital, Dr. Percy Kidd traced the source of the bleeding to ruptured pulmonary aneurysms, while in 3 out of the remaining 5 unruptured aneurysms were discovered. These aneurysms, which are usually the size of a large pea or hazel nut, but which may reach the size of a walnut or even larger, are most commonly found where an artery passes along the wall of a cavity. The vessel wall, which may be softened by tubercular infiltration, bulges on the side next the cavity where it is not supported by lung tissue. Very rarely the vessel may be seen to bulge into the lumen of a bronchus. Occasionally an aneurysm has been found in a caseous patch which is undergoing softening. The aneurysms are usually round and sacculated, but may be fusiform or irregular. Very occasionally they contain laminated

blood-clot. They are commonly single, but not infrequently multiple; as many as thirty have been found of various sizes.

Besides tuberculosis, hæmoptysis, especially in slight amount, may occur in practically all diseases of the lungs. In *bronchiectasis*, hæmoptysis frequently occurs in the later ulcerative stages. Dr. Fowler observed it in 14 out of 35 cases. The hæmorrhage is usually small in quantity, but is sometimes profuse, as much as half a pint of blood may be expectorated. In *carcinoma of the lung* the sputum is frequently blood-stained, and not uncommonly resembles red currant jelly or prune juice, and may rarely contain cancer-cells. In *hydatids* hæmoptysis occurs in the majority of cases. There is usually simply a staining of the sputum, but a severe hæmorrhage may occur prior to rupture of a cyst. Dr. Percy Kidd records one case of fatal hæmoptysis from this cause. In *cirrhosis of the lung*, which may be due to dust inhalation, syphilis, pleuritic extension, or other cause, the slighter degrees of hæmoptysis are not uncommon. According to Dr. Bastian, it occurs in half of all cases. That form of cirrhosis described by Sir Andrew Clark as non-tubercular fibroid phthisis, in which hæmoptysis is not an infrequent symptom, is considered by most modern authorities to be identical with an extremely chronic form of tuberculosis affecting chiefly the supporting framework of the lung, with subsequent fibroid transformation of the tuberculous areas. Hæmoptysis is a rare symptom of *emphysema*, but does occasionally occur and may even prove fatal. It is usually, however, small in amount. It is matter for surprise that it does not occur more frequently considering the frequent association of atheroma of the pulmonary artery with emphysema. In *lobar pneumonia* we have the typical blood-stained, viscid, rusty sputum. Hæmoptysis also occurs in *gangrene*, *pulmonary abscess*, and *actinomycosis*. Streaked expectoration may be observed in cases of *bronchitis*, especially after a paroxysm of coughing.

B. *Diseases of the Heart.* — Hæmoptysis is specially frequent in mitral disease, and more particularly in mitral stenosis. It occurs also in aortic regurgitation. The hæmorrhage is due to rupture of pulmonary capillaries from backward pressure, and is liable to occur after exertion. Large hæmoptysis is not infrequently met with in mitral stenosis. The writer has seen several cases where there have been recurring hæmorrhages exceeding $\frac{1}{4}$-$\frac{1}{2}$ pint in quantity.

C. *Aortic Aneurysm.*—Frequently terminates by rupture into trachea, bronchi, or lung. It seems hardly necessary to state that such hæmoptysis is in the nature of the case rapidly fatal, though there is not uncommonly a pink staining of the sputum for some days or longer due to "weeping" of the aneurysm.

D. *Embolism or Thrombosis of the Pulmonary Artery* causes *pulmonary apoplexy* or hæmorrhagic infarction. The embolus may come from the heart, or may be transmitted from a thrombosed peripheral vein, as in phlegmasia alba dolens. If a fatal termination does not rapidly ensue, there may be fairly profuse hæmoptysis for some considerable time.

E. *Constitutional and Morbid Conditions of the Blood* — *e.g.* Leucocythæmia, purpura hæmorrhagica, scorbutus, hæmophilia, malignant infective fevers. In regard to hæmophilia, some authorities consider that the hæmorrhage is due, not to an alteration in the blood, but to an hereditary structural imperfection of the inner coats of the vessels. A *senile* hæmoptysis has been described occurring in persons over fifty years of age. These may occur and recur in arthritic subjects (Sir Andrew Clark) without any serious disease being present.

F. *Traumatic.*—Blood is expectorated after injuries to thorax and lungs, *e.g.* bullet or sword wounds, or fractured ribs; wounds of larynx and trachea, *e.g.* cut-throat; also after operations on larynx, trachea, or lungs, *e.g.* tracheotomy, paracentesis thoracis (by suction), evacuation of empyema, or pulmonary abscess.

G. *Inflammations and Ulcerations of Larynx, Trachea, and Bronchi*—*e.g.* Tuberculosis, syphilis, carcinoma, foreign bodies, extension of œsophageal epithelioma.

H. *Vicarious.* — Hæmoptysis occurs in rare instances at the menstrual epochs in cases of suppressed menstruation, or at the menopause, or as the result of plethora. It is stated that periodical hæmoptyses have been observed to follow the removal of both ovaries. Sir Thomas Watson mentions a remarkable case of a woman who menstruated through her lungs at each monthly period for forty-two years (from 16 to 58 years of age).

I. *Endemic Hæmoptysis*—due to the presence in the lungs of the Distomum pulmonale—has been described by Drs. Ringer and Manson. Is endemic in *Japan*, *Korea*, and *Formosa*. The disease is characterised by chronic cough with expectoration of a peculiar rusty-brown pneumonic-like sputum. The patient is liable to irregular attacks of hæmoptysis, which may be profuse. In the rusty-brown sputum there are abundant dark-brown, thick-shelled operculated ova. They are oval, smooth, and double outlined, measuring from 80 to 100 μ in length by 40 to 60 μ in breadth. In the course of a month or six weeks a ciliated embryo is developed in each ovum, which, on escaping, at once begins to swim about in the water. The life-history is probably continued in some fresh-water animal, through which it finds its way back to man. Small areas, rather larger than a filbert—the so-called "burrows" — are found scattered throughout the organ, particularly towards the periphery. These "burrows" contain a number of "tunnels" filled with the characteristic rusty-

coloured material, and each containing one or more small distoma. The septa between the tunnels may break down, producing a considerable cavity.

The distomum pulmonale v. Ringeri v. Westermanni is reddish brown in colour, oval in form, and so thick and fleshy that it is almost circular in transverse section. It measures 8 to 10 mm. by 4 to 6 mm., and is covered by minute spines.

Exciting Causes of Hæmoptysis.—Very often there is no exciting cause, but an attack is sometimes determined by muscular exertion, a paroxysm of coughing, mental agitation, menstruation, straining at stool, alcoholism, or extremes of heat and cold.

Pathological Appearances.—In the case of a fatal hæmoptysis the mucous membrane of the air-passages is usually blood-stained. Blood is found in the trachea, bronchi, and lungs. There may be a thick cylindrical clot extending from the trachea down to the smaller bronchi. The blood is as a rule most abundant in the vicinity of the ruptured vessel, though it is often present in parts quite remote, and not infrequently a large amount of blood-clot may be found in the opposite lung. This flooding of the sound lung with blood is commonly the cause of death in fatal cases by asphyxia. Blood may even be found in the upper lobe of the opposite lung, showing that the inspiratory efforts have been powerful enough to force the blood up against the action of gravity. As regards the heart, it presents the appearances characteristic of death from asphyxia or syncope. In cases which recover, the effused blood is usually completely absorbed; but Dr. Reginald Thomson has shown that in a number of cases the relics of blood are to be found in the presence of hard, fibrinous, pigmented nodules. In cases of fatal hæmoptysis due to pulmonary embolism we find large hæmorrhagic infarctions. In cases which recover, these alter into hard, wedge-shaped fibrinous masses.

Putrefactive changes very rarely occur in the effused blood except in those cases where the hæmorrhage is the result of inhalation of blood from the parts above the larynx (*e.g.* epistaxis and mouth operations), when it is due to the presence of the accompanying putrefactive organisms.[1]

[1] *Phthisis ab Hæmoptœ.* — Hæmoptysis was such a common and striking feature of consumption that the older writers, from Hippocrates downwards, erroneously regarded phthisis itself as directly due to infective and putrefactive changes in the effused blood. In later times Niemeyer himself reverted to this old view, basing his opinion on the fact that sometimes hæmoptysis is really the first symptom, and also on the fact that pyrexia not uncommonly follows an hæmoptysis. Since, however, the discovery by Koch of the pathogenic tubercle bacillus it has been discovered that this after-pyrexia described by Niemeyer is in reality due to infective tuberculous broncho-pneumonia of other portions of the lungs. The term is therefore a misnomer.

Symptomatology.—The onset of an hæmoptysis may be sudden or preceded by one or two days of staining. Occasionally there is a preceding sense of tightness across the chest. There is usually in tubercular cases a previous history of cough and expectoration, though in about 3 per cent of cases hæmoptysis is actually the first symptom. It is to be noted that the onset is not as a rule associated with exertion or strain, as one might suppose. The patient simply experiences a tickling sensation about the fauces, has a saltish taste in the mouth, and suddenly discovers that his mouth is full of bright blood. The amount expectorated varies in quantity from a mere streak or staining of the sputum to one, two, or more pints of blood. In early phthisis the bleeding is usually small in amount, but is liable to recur. Profuse hæmoptysis generally occurs in the later stages. In Dr. Pollock's return of 341 cases of large hæmoptysis 12 per cent occurred in the first stage, 41 per cent in the second, and 46 per cent in the third. The blood is generally bright red and frothy, but when very profuse is often dark and clotted, especially if it has gradually accumulated in pulmonary cavities or bronchial tubes. When the flow is not excessive the blood is often mixed with sputum. Blood-casts of the smaller bronchi are occasionally expectorated. Usually the expectoration remains blood-stained for some days after, or there are brownish-black specks in the sputum.

In the case of a profuse hæmoptysis the patient is generally greatly alarmed—the face is pallid and bedewed with sweat; there is faintness, feeble pulse, and coldness of extremities. The blood is brought up with a frequent short cough, and the patient may continue to bring it up for some hours. In severe cases blood may pour out at the mouth and nose, and may rapidly fill a fairly large porringer (or small basin). Some of the blood is often swallowed, and is either vomited or passed per rectum. Owing to the collapsed condition of the patient the temperature becomes subnormal. When the shock has passed off the temperature rises generally within twelve hours to normal or to its original height before the hæmorrhage occurred. In a few instances a high temperature may be observed for some time afterwards, due probably to the formation of foci of infective broncho-pneumonia, the result of insufflation of the lungs with blood charged with micro-organisms from the cavities and softened areas. Dr. Williams regards this after-pyrexia as of ill omen. It may be some time before the patient recovers from the mental depression and from the ever-present dread of its recurrence. The majority of all cases do tend to recur. The term *hæmorrhagic phthisis* has been applied to those cases where hæmorrhage is a constant and frequent phenomenon. This frequent recurrence may be explained by the supposition that the

rent in the wall of the artery or aneurysm has only undergone partial repair.

It may be here noted that it is quite possible for a fatal hæmorrhage to occur into a large cavity without any blood being expectorated at all.

As regards the *examination of the chest* it is very important to remember that for some time after a severe hæmoptysis the patient should be disturbed as little as possible. He should not be asked to take a deep breath or to cough or speak, and the chest should on no account be percussed. The lungs may be auscultated during tranquil respiration without risk. In addition to the signs of the accompanying lung disease one can often hear fine moist crepitant râles over the area of effusion, and, according to Laennec, these are frequently audible in the vicinity of (just above) the roots of the lungs. These may persist for some days, or even weeks.

Differential Diagnosis.—A. *Differentiation of the various causal Conditions that lead to Hæmoptysis.*—They are as a rule readily distinguished by their associated symptoms and clinical features. Owing to its extremely frequent occurrence in connection with phthisis the presumption in the absence of heart disease is always in favour of tuberculosis, even although no physical signs of pulmonary disease can be detected. On the other hand, if a presystolic murmur is heard at the cardiac apex we can generally dismiss all idea of phthisis, as mitral stenosis and pulmonary tuberculosis very rarely coexist. The following points may also be noted:—The sputum in acute pneumonia has the well-known rusty colour; in gangrene it frequently resembles prune juice; in carcinoma it has been likened to red currant jelly; in pulmonary œdema it consists of a sero-sanguineous fluid; in brown induration of the lung, and in a pulmonary apoplexy of some standing, it may be of a dark brown colour, owing to the presence of hæmatoidin crystals.

B. *Diagnosis from other Hæmorrhages.*—(1) *Hæmatemesis.*—In hæmatemesis there is usually a history of dyspepsia, and there is often accompanying pain in the epigastrium. The blood is acid in reaction from admixture with gastric juice; it is dark in colour, and more or less altered by the gastric juice. It is preceded by nausea, and comes up with vomiting, and may be mixed with food. The blood is brought up all at once, and not in successive mouthfuls; faintness often precedes the hæmorrhage; subsequent coffee-ground vomit and tarry motions. In hæmoptysis, on the other hand, there is often an antecedent history of cough or other pulmonary symptoms. The blood comes up with coughing. It is alkaline in reaction, is bright red and frothy, and mixed with mucus. There are often air-bubbles in the coagulum; there are occasionally blood moulds of the smaller bronchi. It is preceded often by a tickling sensation in the throat; is generally followed for some days by stained sputum (this is probably the most important diagnostic point); the hæmoptysis is generally repeated; microscopic examination may reveal tubercle bacilli, elastic fibres, or pulmonary epithelium. (In primary hæmoptysis the expectorated blood should always be stained for bacilli.)

The following points must be noted in order to guard against possible fallacies:—The act of vomiting may excite coughing, and *vice versa*; there may be no cough at the first appearance of hæmoptysis; blood from the lungs may be swallowed, producing secondary hæmatemesis and melæna; and hæmatemesis may occur from rupture of varicose veins at the lower end of œsophagus as a symptom of hepatic cirrhosis.

(2) *Epistaxis.*—In nasal hæmorrhage blood may pass back into the pharynx and be expectorated. Anterior and posterior rhinoscopy may be necessary to decide whether the hæmorrhage arises in the nose or posterior nasal space. Hæmoptysis has been known to occur in connection with undue vascularity of the inferior turbinated bones without epistaxis.

(3) *Hæmorrhage from Throat or back of Tongue.*—Hæmoptysis to any extent from this cause is very rare. Streaks of blood are occasionally observed in the expectoration accompanying some minor diseases of the pharynx. Sometimes the presence of blood in the mouth on rising from sleep has been observed in varix of the tongue, mouth, and pharynx. In this connection it may be noted that real hæmoptysis in childhood is rare, even in cases of pulmonary tuberculosis. When a young child does spit up blood it is generally owing to the violence of the coughing, as in the paroxysms of whooping-cough, and often comes from the throat, nose, or gums, and occasionally from an ulcer of the frenum linguæ.

(4) *Spurious Hæmoptysis and Malingering.*—Neurotic anæmic girls often speak of finding blood on their pillows on waking, or of their mouths being filled with blood in the morning. They acquire the habit of sucking their cheek or gums. The latter are seen to be pale, spongy, and exuding blood. The sputum in these cases is generally, according to Dr. Wagner, of a pale red colour, not so bright as in ordinary hæmoptysis, and on settling sometimes presents a reddish-brown sediment. The blood is mixed with saliva, but contains no cylindrical or ciliated epithelium. The blood only appears in the morning, and there are no physical signs in the lungs.

Malingerers may produce blood in the same way, or may actually add blood derived from another source. The late Professor Hughes Bennett discovered a case of malingering by examination of the blood with the microscope. The corpuscles were oval instead of round, thus

proving that they did not come from a mammalian source at all.

PROGNOSIS.—Hæmoptysis is very rarely fatal. In the large proportion of cases there is a tendency to spontaneous arrest of the hæmorrhage. Dr. Samuel West states that the mortality from hæmoptysis in phthisis is less than 3 per cent. Sometimes an hæmoptysis occurs without warning, continues for a few days, and then disappears, leaving no traces behind, and no physical signs develop. In all probability, however, there has really been in these cases a tubercular focus, which has cicatrised and healed up. Such foci at one or other apex are frequently found post-mortem. Of 4000 cases of hæmoptysis observed at Brompton Hospital the quantity in 69 per cent was under ℥ss. Still profuse and repeated hæmoptysis must always be regarded as a grave condition. The large majority of fatal cases occur in the stage of excavation, and are due to the rupture of a pulmonary aneurysm. Fatal hæmoptysis is more common in males than in females, and is rare in the case of children and old people, though the writer has seen in the Royal Chest Hospital a fatal termination in the case of a child under three years of age. Dr. Reginald Thomson says that fatal hæmoptysis is more common in the summer months—June and July. Death is due, in the majority of cases, to asphyxia from regurgitation of blood into the bronchial tubes, more particularly of the sound lung. The patient is practically drowned in his own blood. In a few cases death is due to syncope.

As regards the influence of hæmoptysis on the progress of pulmonary tuberculosis opinions vary somewhat. The laity very often regard spitting of blood in quantity as "the beginning of the end." There is certainly no clinical evidence in support of such a view. Dr. Pollock, in his *Elements of Prognosis in Phthisis*, says hæmoptysis, even if profuse, may have no appreciable effect on the subsequent health of the individual. In not a few cases the effects seem actually beneficial. It is easy to understand that this is the case in hæmoptysis due to chronic pulmonary congestion by acting as a safety-valve. May it not occasionally act in a similar way in the acute pulmonary hyperæmias of pneumonia and early tuberculosis? Certainly one frequently sees cases of recurring hæmoptysis with practically no chest symptoms and little if any deterioration of the general health. Dr. Williams, after careful observation, expresses the opinion that the influence of hæmoptysis in the first stage is nil, whereas hæmoptysis occurring in the second and third stages curtails the average duration of the disease.

It must also be remembered that a certain proportion of cases of chronic phthisis may assume a subacute progressive character after an hæmoptysis has occurred. This, as already explained, is probably due to the onset of secondary infective broncho-pneumonia.

TREATMENT.—The indications are twofold:—A. *Promote thrombosis*, which is the natural process of repair, by lowering the blood-pressure in the pulmonary artery. In this way we arrest the bleeding. B. *Prevent as far as possible the regurgitation of blood* into the neighbouring bronchi, and more particularly into the bronchus leading to the opposite lung; and *promote its expulsion therefrom in the event of its occurrence.* In this way we generally minimise the immediate danger of fatal asphyxia, and also prevent the later occurrence of infective broncho-pneumonia.

A. Thrombosis is promoted by the following means:—(1) *Rest*—Fulfils all therapeutic indications, and is much the most important of the cardiac sedatives. Nothing more is required in mild cases. The syncopal condition of the patient also acts as an excellent cardiac depressant. Rest ought to be as absolute as possible—general, local (cardiac and respiratory), and mental. The patient is confined to bed in a cool, airy room, is kept quiet and undisturbed, is lightly clad, in severe cases is forbidden to speak above a whisper, is only asked essential questions; the cough is checked by sucking pieces of ice or by a sedative linctus. Dr. Mitchell Bruce lays stress on the mental aspect of the question, and makes a point of reassuring the patient by a few encouraging words. He is requested to lie as still as possible. In the European sanatoria *ligature of the extremities* is frequently carried out with the view of diminishing the influx of blood into the lungs, and so checking the hæmorrhage. A special apparatus is employed, but the same effect can be produced by a tight bandaging of the upper arms and thighs.

(2) *Opium*—Is by far the most important drug in the treatment of hæmoptysis. It acts as a cardiac and respiratory sedative and depressant, checks the cough, allays the general restlessness, soothes the mental distress and agitation, and promotes sleep. The best method of administration is by a *subcutaneous injection of morphia* (gr. $\frac{1}{4}$-gr. $\frac{1}{3}$), which may have to be repeated once or even twice. Some physicians recommend a combined injection of morphia (gr. $\frac{1}{4}$) and atropine (gr. $\frac{1}{100}$).

(3) *Low Diet.*—Nothing is given for some hours except small pieces of ice to suck at considerable intervals, so as to prevent flatulence with its accompanying restlessness. The sucking of ice relieves the thirst as well as the cough, and likewise calms the patient's anxiety by giving him the feeling that something definite is being done for his relief. After a little time iced milk is given in tablespoonful doses and gradually increased in quantity. Everything must be cold. Cold milk, cold meat jelly or essence, with a little bread and butter,

is usually sufficient for the first few days, along with, it may be, a little pounded meat and milk pudding. Alcoholic stimulants must be strictly avoided, except in the rare instances where there is danger of syncope. The pulse must be carefully watched.

(4) *Saline Purgatives.*—These lower the blood-pressure by acting as derivatives. Dr. Kingston Fowler recommends one drachm of sulphate of magnesia with 20 grains of sulphate of soda given every four hours.

(5) *Ice-bag to Chest*—Is now little used. Has probably no effect on the ruptured vessels, but if applied over the præcordial region may have some effect in lessening the cardiac excitability.

(6) *Other Medicinal Remedies.*—(*a*) *Astringents.*—Though used extensively by the older school of practitioners, their value is exceedingly doubtful. Ergot, which is undoubtedly a valuable drug in uterine hæmorrhage, has the greatest reputation, while hazeline, gallic acid, and turpentine are also employed. Sir R. Douglas Powell speaks well of acetate of lead in combination with dilute sulphuric acid. Hydrastis Canadensis is used by some American physicians. It is not easy to understand what effect such astringents can possibly have on an ulcerated vessel or ruptured aneurysm. Indeed, Bradford and Dean have shown that ergot and hazeline actually raise the blood-pressure in the pulmonary circulation. Clinical experience is also against the use of these remedies. During the writer's term of office as house physician at Brompton Hospital he was never once called upon to administer ergot or any other astringent. They are better withheld altogether, as they derange digestion and induce constipation. (*b*) *Aconite* has been recommended by Dr. Andrew on the ground that it lowers the blood-pressure in the pulmonary circulation (Bradford and Dean).

B. The prevention of the regurgitation of blood and its subsequent expulsion in the event of its occurrence are secured by the following means:—

(1) *Position of the Patient.*—This is of the utmost importance. The patient, on the occurrence of a severe hæmoptysis, should be immediately turned and made to lie on the affected or most affected side. It is the corresponding lung that is the source of the hæmorrhage. If this is done, then any regurgitation of blood that may occur will, owing to gravity, take place from the trachea into the bronchus of the affected lung, where it will do little harm, as that lung is largely functionless, especially in the stage of excavation, when profuse hæmoptysis is so common. On the other hand, if, on the incidence of the hæmoptysis, the patient happens to be lying on his sound side, the blood will regurgitate from the trachea into the bronchus leading down to the healthy functioning lung, and breathing becomes almost impossible. If the patient be lying on his back the blood will regurgitate into both lungs. In the writer's opinion, flooding of the sound lung with regurgitated blood is the most common cause of fatal asphyxia, a result which would often be avoided were this rule always attended to. The writer, while house physician at Brompton Hospital,[1] was in the habit of writing the letters R. or L., as the case might be, on the bed cards of all the cases of phthisis to indicate the affected or most affected side. The charge nurses had instructions, in the event of a hæmoptysis, to immediately turn the patient on the affected side. In spite of the fact that there were numerous cases of profuse hæmoptysis, not a single fatal termination occurred during his term of office. The patient's friends should be told of the importance of position.

(2) *Morphia*, by checking the cough, soothing the restlessness, and calming the mental distress, does much to diminish the frequent inspiratory efforts which conduce to regurgitation.

(3) *Artificial Respiration.*—If there is threatened asphyxia from flooding of the lungs with regurgitated blood, it is advisable at once to have recourse to some form of artificial respiration, with the patient lying on the affected side. Such a case occurred at Brompton Hospital, where, by the use of Sylvester's method of artificial respiration, the writer succeeded in getting most of the blood ejected, with consequent speedy recovery, and this too after the patient's condition had been pronounced hopeless by the physician in charge who happened to be present when the hæmoptysis occurred.

(4) *Stimulating Expectorants.*—After a day or two it is often advisable to give small doses of ipecacuanha, squills, or ammonium carbonate, in order to assist in the expulsion of the small relics of blood from the minuter bronchi. There is thus less likelihood of a subsequent infective broncho-pneumonia.

After-treatment and Prophylaxis.—Diet should be sparing, and alcoholic stimulants avoided for some weeks. The patient may be allowed to get up after the sputum has been free from staining for a few days. Should take occasional laxatives and carefully avoid all excessive exertion. Ferruginous remedies and tonics may be necessary to counteract the anæmia and associated debility. Primary hæmoptysis is a warning of incipient lung disease, and therefore a change of climate is indicated as soon as the patient can bear the journey. Traube spoke highly of the value of respiratory exercises as a preventive measure in cases of chronic recurring hæmoptysis associated with congestion of the respiratory organs.

[1] Brompton Hospital is the largest consumption hospital in Great Britain. There is provision for 350 patients, of which 80 per cent are cases of pulmonary tuberculosis.

Hæmorrhage.

See also Abortion (*Symptoms*); Adrenal Glands (*Adrenalin, Uses*); Anæmia (*Causes*); Anæmia, Pernicious (*Symptoms, Morbid Anatomy*); Appendix, Appendicitis (*Results of Infection*); Arteries, Injuries of (*Wounds*); Astringents; Bladder, Injuries of (*Penetrating Wounds*); Brain, Affections of Blood-Vessels; Brain, Surgery of (*Intracranial Hæmorrhage*); Brain, Cerebellum, Affections of (*Hæmorrhage*); Bronchi, Bronchial Glands (*Morbid Anatomy and Pathology, Pressure*); Cautery (*Red Heat, Uses*); Chlorosis (*Diagnosis*); Curettage, Uterine (*Indications*); Embolism; Enemata (*Saline, Uses*); First Aid (*Sundry Accidents, Hæmorrhage*); Gall-Bladder and Bile Ducts, Diseases of (*Injuries, Ulceration and Sequelæ, Hæmorrhage*); Hæmatemesis; Hæmaturia; Hæmoglobinuria; Hæmophilia; Hæmoptysis; Heredity (*Hæmophilia*); Hysteria (*Digestive Disorders, Hæmatemesis*); Intestines, Diseases of (*Tuberculous Ulcers*); Intestines, Surgical Affections of (*Obstruction, Diagnosis, Intussusception*); Kidney, Surgical Affections of (*Stone, Symptoms*); Labour in Multiple Pregnancy (*Management, Third Stage, Hæmorrhage*); Labour, Post-Partum Hæmorrhage; Labour, Injuries to the Generative Organs; Labour, Operations (*Cæsarean Section*); Larynx, Acute and Chronic Inflammations (*Laryngeal Hæmorrhage*); Larynx, Malignant Disease of (*Symptoms*); Leucocythæmia (*Symptoms*); Leucocytosis (*Post-Hæmorrhagic*); Liver, Diseases of (*Cirrhosis, Symptoms; Acute Yellow Atrophy, Symptoms*); Lungs, Vascular Disorders; Malaria (*Sequelæ*); Mammary Gland, Diseases (*Inflammatory Affections, Mastitis of Adolescence, Vicarious Hæmorrhages*); Mediastinum (*Growths, Diagnosis, Hæmorrhage into Mediastinum*); Medicine, Forensic (*Wounds*); Melæna; Meninges of the Cerebrum (*Vascular Disturbances*); Menstruation and its Disorders (*Menorrhagia and Metrorrhagia*); Muscles, Diseases of (*Vascular Disturbances*); Nails, Affections of (*Anomalous, Hæmorrhages into Nail Substance*); Nephritis; New-Born Infant (*Diseases, Buhl's Disease, etc.*); Obesity (*Adiposis Dolorosa, Symptoms*); Œsophagus (*Wounds, Inflammation*); Pancreas, Diseases of (*Hæmorrhage*); Paralysis (*Acute Anterior Poliomyelitis, Hæmorrhage into Spinal Cord*); Pelvis, Hæmatocele and Hæmatoma; Pharynx, Examination of (*Hæmorrhages*); Pregnancy, Pathology, Ovum and Decidua (*Affections of Decidua*); Pregnancy, Diseases of Placenta and Cord (*Hæmorrhages*); Pregnancy, Affections of Generative Organs (*Cancer of Uterus*); Pregnancy, Hæmorrhage (*Antepartum, Varieties*); Pulse (*Frequency, Significance*); Retina and Optic Nerve (*Retina, Hæmorrhages*); Rheumatism, Rheumatoid Arthritis (*Clinical Characters, Subacute and Chronic Varieties*); Scarlet Fever (*Complications, Hæmaturia*); Scurvy (*Infantile*); Scurvy (*in Adults*); Smallpox (*Clinical Variations, Variola Hæmorrhagica*); Spinal Cord, Medical (*Morbid Anatomy, Vascular Lesions*); Stomach and Duodenum, Diseases of (*Morbid Anatomy, Ulceration; Special Symptomatology, Ulcer, Cancer*); Teeth (*Extraction, Hæmorrhage*); Temperature (*Alterations, Depression, Causes*); Typhoid Fever (*Complications and Sequelæ, Hæmorrhages, Various*); Umbilicus, Diseases of (*Hæmorrhage*); Uterus, Malformations of (*Atresia Vaginæ, Treatment, Hæmorrhage during*); Uterus, Displacements of (*Retroversion, Symptoms*); Uterus, Inflammations of (*Endometritis, Symptoms*); Uterus, Non-Malignant Tumours (*Symptoms*); Uterus, Malignant Tumours of (*Symptoms*); Vitreous Humour, Diseases of (*Hæmorrhage*); Whooping-Cough (*Complications and Sequelæ, Hæmorrhages*); Yellow Fever (*Clinical History, Hæmorrhages*).

Hæmorrhage is always present when a blood-vessel is wounded, and the loss of blood may be so trivial as to give rise to no definite constitutional effect, or so severe in case of wound of one of the largest arteries as to prove rapidly fatal. The blood may escape externally through an open wound, or may become extravasated subcutaneously, or into one of the great cavities of the body. A patient may die from hæmorrhage without one drop of blood being shed external to the body, *e.g.* intraperitoneal hæmorrhage. The main considerations in estimating the danger of a severe hæmorrhage are the quantity of blood lost by the individual and the rapidity with which the hæmorrhage occurs. Of all surgical emergencies there is none more urgent, and none which makes greater demand upon the judgment and presence of mind of the surgeon than a rapid and severe hæmorrhage, and upon its proper treatment the life of the patient entirely depends.

It is customary to describe four varieties of hæmorrhage, classified according to the source of the hæmorrhage, viz. arterial, venous, capillary, and parenchymatous. In addition to the above we distinguish, according to the time of the occurrence of the bleeding, (*a*) primary; (*b*) reactionary, intermediary or consecutive; and (*c*) secondary hæmorrhage.

1. Arterial Hæmorrhage.—In this variety the blood is bright red (oxygenated), and escapes in jets from the wounded artery synchronously with the beats of the heart. It is to be borne

in mind, however, that in patients who are deeply anæsthetised or suffering from partial asphyxia the blood escaping from a wounded artery may be dark-coloured.

2. Venous hæmorrhage is characterised by the dark colour of the blood and by its flowing in a continuous stream, due to the absence in the veins of the impulse of the heart-beat. In general the walls of the vein collapse.

3. Capillary hæmorrhage is characterised by the oozing of blood from several points of the wounded surface. The amount of blood lost at each bleeding point is but small, and it is only rarely when this variety of hæmorrhage persists for several hours that the amount of blood lost is sufficiently considerable to place life in danger. Owing to the fact that the capillaries in the mucous membranes are larger and more abundant than those of the skin, hæmorrhages from the former are apt to be more troublesome than from cutaneous wounds.

4. Parenchymatous hæmorrhage is chiefly characterised by its occurrence in parts or organs which present structural peculiarities, for example in erectile tissue, where little arteries open directly into veins without the intermediation of capillaries, as in the erectile tissue of the genitalia and in the spleen. It is also to be observed in parts whose normal vascular condition is altered, particularly by thrombosis and malignant infiltration.

A. Primary hæmorrhage occurs immediately upon receipt of the injury to the vessel. It may, of course, present the characteristics of any of the above-described anatomical varieties of hæmorrhage.

B. Reactionary, intermediary, or consecutive hæmorrhage occurs after the establishment of the period of reaction, *i.e.* usually within the first twenty-four hours after the receipt of an injury or after a surgical operation. It is due generally either to the slipping of a ligature or to the increasing blood-pressure forcing out of the injured vessels the coagula which plug their divided ends.

C. Secondary hæmorrhage occurs after the expiration of at least twenty-four hours from the time of the injury. It is most frequently observed from the sixth to the fifteenth day. It usually occurs in a suppurating wound, and is caused by ulceration of the walls of the vessel, the result of a septic arteritis; very rarely is it due in an aseptic wound to the too rapid absorption of a catgut ligature or to incomplete repair in the injured artery. Secondary hæmorrhage, in olden days a frequent and very serious occurrence, has now, thanks to surgical cleanliness, become a rare wound complication.

Constitutional Symptoms of Hæmorrhage.—A patient suffering from severe hæmorrhage presents the following appearance. He is pallid, cold, and yet beads of perspiration abound, the lips are blanched, but show a faint lividity, the pulse feeble, quick, and small, and perceptible only perhaps in the large arteries. The breathing is irregular and quickened, the alæ nasi dilate, and complaint is made of shortness of breath, the temperature is below normal, convulsive movements, marked restlessness, and vomiting may occur. There are generally abnormal sensations of sight and hearing, as flashes of light, a sound as of rushing water, or buzzing in the ears, and in extreme cases syncope, unconsciousness, and death may supervene.

Whilst the quantity of blood lost and the rapidity with which the hæmorrhage occurs are, as above stated, the main considerations, other factors have to be taken into account; thus aged persons, and very young children, stand the loss of blood badly, but they differ markedly in their recuperative power—children recovering in cases not rapidly fatal quickly and thoroughly, aged people much more slowly, and indeed rarely completely.

Women at the period of parturition recover from hæmorrhages which at any other time would almost certainly prove fatal.

Hæmorrhagic Fever.—After a serious hæmorrhage the patient frequently presents the condition known as hæmorrhagic fever, in which are found acceleration and sometimes irregularity of the pulse-rate, elevation of temperature, extreme thirst, diminution of the quantity of urine passed per diem, and low muttering quiet delirium. This condition, formerly held to be due to septic absorption, is now generally believed to be in great part due to insufficient supply of blood to the great nerve-centres.

Spontaneous Arrest of Hæmorrhage.—Fortunately in the great majority of cases natural processes stay the blood flow before the hæmorrhage proves fatal. If an artery be divided completely, it, owing to its elasticity, retracts, and its muscular coat contracting narrows or even closes the open wound in the vessel. In this way the loss of blood is lessened or arrested, and coagulation at the seat of injury is favoured. In small arteries muscular contraction may suffice of itself to completely arrest the hæmorrhage. Cardiac syncope not infrequently, by diminishing the pressure of the blood in the artery, acts as a potent factor in arresting hæmorrhage. Coagulation of the blood is also aided by the exposure of the blood to atmospheric influences, and to the curling in towards the lumen of the vessel of its middle and internal coats. All the above factors aid in bringing about the formation of a blood-clot, which lies inside the sheath of the vessel, and is applied to the end of the divided artery, constituting the "temporary external coagulum." A second blood coagulum now forms within the lumen of the severed vessel. This, which is known as the "internal coagulum," frequently extends upwards as far as the next large branch of the vessel. The external coagulum becomes in

course of time entirely absorbed, whilst as the result of the subsequent organisation of the internal coagulum the artery becomes completely obliterated at the seat of injury. In lacerated wounds of arteries the middle and internal coats curl upwards and inwards into the lumen of the vessel, and the external coat and sheath become twisted over the opening. Hence the fact that lacerated wounds of vessels scarcely bleed appreciably. In wounds of veins retraction and contraction are not so perceptible as in the case of divided arteries, but the collapse of the wall of the vein greatly aids the formation of a coagulum.

Diagnosis of Hæmorrhage.—When the blood escapes externally no difficulty in making a diagnosis of hæmorrhage can occur. Very different, however, is the diagnosis of concealed hæmorrhage, as when the blood finds its way into one of the great cavities of the body, the peritoneal cavity for example. In such cases, though most of the constitutional symptoms of severe hæmorrhage be present, they frequently are only by the employment of the greatest care able to be diagnosticated from those of shock. In the great majority of cases the physical signs enable a correct diagnosis to be made.

Treatment of Hæmorrhage.—Constitutional treatment is called for after a severe hæmorrhage (the bleeding having been, if possible, first arrested). Owing to the general weak state of the patient energetic treatment is often called for. The head should be placed as low as possible to prevent anæmia of the brain, and the patient be kept absolutely at rest, the windows of the room should be opened to ensure a free supply of fresh air, the patient should be warmly covered with blankets. The arms and the legs may be elevated, or better, encircled in Esmarch's rubber bandages, in order that blood may be driven out of them into the brain and the thoracic viscera. Large quantities of warm fluids may be given to the patient to drink, or better, a copious enema of water, 1 to $1\frac{1}{2}$ pints, may be injected into the large bowel, and will generally be retained. Heart stimulants, such as strychnine, carbonate of ammonia, or better, hypodermic injections of ether or brandy, are very beneficial. There is, however, a danger of producing over-stimulation, so that these remedies need to be judiciously administered. In very extreme anæmia from hæmorrhage, when the above-mentioned means fail, there still remains one remedy which has often saved life in the most desperate cases, and which should never be omitted, viz. the transfusion of a sterilised ·07 per cent of sodium chloride (see "Transfusion").

It may be well here to point out that the cause of death from hæmorrhage is dependent solely upon mechanical conditions. Death occurs when the blood is insufficient in amount duly to satisfy the capacity of the vascular system. The heart is thus like an empty pump, and is unable to drive on the blood. Here the obvious indication is to increase the volume of blood by the addition to it of some liquid, preferably normal salt solution, in order to enable the circulation to be carried on.

In place of intravenous injection two or three pints of normal saline solution may be injected into the subcutaneous tissue, preferably under the breasts. Rectal injection of warm saline solution is also an invaluable method, owing to the rapidity with which absorption of fluid takes place in the large intestine. In cases of severe hæmorrhage 1 to 2 pints of warm water injected into the rectum will be retained and rapidly absorbed. This method requires no assistants or sterilisation, and is only second in rapidity of action to intravenous injection.

Local Treatment of Hæmorrhage.—Primary Hæmorrhage.—Before describing the various measures which may be employed in order to control, either temporarily or permanently, bleeding from a wound, it is well to emphasise the fact that pressure properly applied to the bleeding point will suffice to stop temporarily bleeding even from the largest vessels whilst measures for the permanent arrest of the hæmorrhage are being undertaken. This is a point too often lost sight of.

1. *Position.*—Elevation of the arm or leg always diminishes and may of itself suffice to arrest hæmorrhage from the part in question. Forced flexion of a joint, elbow or hip, for example, on the proximal side of the bleeding point has a similar effect.

2. *Compression.*—This may be applied directly to the bleeding point, preferably by the fingers of the surgeon or an assistant, or the main artery may be compressed between the wound and the heart.

(*a*) *Digital Compression.*—As mentioned above, digital compression will temporarily arrest bleeding from any vessel, but can only be kept up for a comparatively short time.

(*b*) *Direct Pressure by Means of Antiseptic Compresses.*—This is a valuable method of treatment in cases where there is considerable oozing of blood from the wound, particularly in the case of a large cavity. The wound may be tightly stuffed with antiseptic gauze or wool, and over this a bandage, preferably elastic, should be somewhat firmly applied. The gauze packing should be left in position for two or three days, though if the bleeding be arrested the bandage may be removed earlier. This method is not to be recommended in the case of furious bleeding from a large artery.

(*c*) *Tourniquets* are instruments by means of which pressure is applied to the main artery on the proximal side of the wound. Their employment is an invaluable method of securing temporary control of arterial hæmorrhage, but

cannot be continued for more than one to two hours owing to the extreme pain they cause.

Improvised Tourniquet.—Fold a handkerchief into a band 1 to 2½ inches thick; into the middle of this place a round stone or large round bullet, tie the two ends of the handkerchief loosely together after encircling the limb in such a way that the stone is placed over the main artery. Now insert a strong stick inside the encircling band of handkerchief and twist the stick until hæmorrhage ceases. This is a simple and effective emergency tourniquet.

Elastic Constriction (*Esmarch*).—An elastic band or cord forms the most useful tourniquet, as by its use the circulation in a limb can be entirely arrested. It is made to encircle the limb above the bleeding point. It should be applied with just sufficient firmness to control the hæmorrhage. If too tightly applied motor paralysis and laceration of muscles may be caused. An objection to this tourniquet is the fact that free capillary bleeding due to vaso-motor paralysis generally follows the removal of the constriction. The elastic band is greatly to be preferred to the cord-like or tubular constrictor.

Among other forms of tourniquet mention need be made only of two, Petit's and Lister's.

3. *Cold and Hot Water.* — Cold water just above the freezing point (32° F.) temporarily checks bleeding, but is considerably inferior to hot water. It seems unquestionably to increase the shock, and for this reason is inadvisable in severe hæmorrhage. Hot water which has been sterilised by boiling, and then allowed to cool down to 130° to 120° F., is an excellent styptic for considerable oozing from large surfaces or cavities, though it is useless when the hæmorrhage comes from a large artery. By the employment of compresses soaked in water at 120° to 130° F. pressure and hæmostasis are at the same time obtained. Hot water seems probably, in great part by supplying heat, to diminish shock, and is therefore doubly indicated.

4. *Styptics*, as liquor ferri perchloridi, charpie, cobwebs, etc., were greatly used in olden days. Their employment is always inadvisable, and we mention them merely to condemn them as harmful and unscientific and dirty.

5. *Torsion* is a valuable and efficient method of arresting hæmorrhage in arteries which have been completely divided. The bleeding vessel is clamped with forceps, and a second forceps seizes the artery a short distance higher up at right angles to the vessel. The second pair of forceps holds securely the artery whilst a few twists of the first forceps in the direction of the long axis of the vessel suffice securely to obliterate the lumen of the artery.

6. *Acupressure.*—In this method a needle is passed under the vessel from the skin surface, which is twice pierced, and a figure-of-eight loop of silk is passed over the vessel around the two ends of the needle so as to exert compression. A more rapid method is to introduce the needle parallel with the artery, and then turning the needle through an angle of 90 degrees, pass it under the artery, which in this way suffers some degree of torsion as well as of compression.

7. *Suturing.*—Suture of a wound in an artery is not to be recommended owing to the tension in the vessel. It is a useful method, however, in wounds of veins. The suture should not penetrate the internal coat.

8. *Forcipressure.*—This is perhaps the most valuable method we possess, especially when used in conjunction with ligature of the larger vessels in the wound. Forcipressure is used in almost every surgical operation. Every bleeding point is clamped with artery forceps, which themselves suffice to arrest the bleeding in the case of tiny vessels. Any vessel, however, of any size requires to be ligatured or twisted. In certain instances in which, for various reasons, it is impossible to ligature a bleeding point, the vessel may be clamped and the forceps may be allowed to remain from two to four days, when the bleeding point will, except in very large arteries, be safely sealed.

9. *Cauterisation*, applied by the employment of Paquelin's cautery, the electric cautery, or any hot iron, is in certain cases a valuable means of arresting hæmorrhage, provided that the cautery be kept at a dull cherry-red heat. If used at a white heat the hæmostatic effect is lost. A disadvantage in cauterisation is the necessary production of a slough. The method, however, is valuable in not a few cases in which a ligature cannot be applied.

10. *Ligature.*—(*a*) With inclusion of surrounding tissues in certain cases in which, for any reason, *e.g.* the density of the tissues or the unhealthy condition of the vessel wall, the bleeding end of an artery cannot be clamped and ligated. A curved needle threaded with a ligature is then to be carried to some depth into the tissues around the bleeding vessel, which is sufficiently compressed by tying the ligature.

(*b*) *Ligature proper of the Vessel freed from surrounding Tissues.*—This is the method generally employed. Silk and chromic catgut are the materials which are generally to be recommended. Every substance employed as a ligature must be carefully sterilised. Silk can readily, by means of boiling for a few minutes, be rendered sterile, and this is its great advantage. Catgut, being made from the submucosa of the intestine of the sheep, is naturally most septic. It requires soaking for days in a strong antiseptic solution to ensure it being germ free. A great advantage of chromic catgut lies in the fact that it is absorbed in from one to three weeks. Thick catgut, however, is difficult to sterilise.

In applying a ligature the mouth of the artery should be clamped with forceps and drawn out somewhat from its sheath, and the artery tied with a reef knot.

It is safer in every instance to tie the ligature with sufficient force to ensure rupture of the middle and internal coats of the vessel. When an artery is completely divided, both the proximal and distal ends require ligation. Should an artery be incompletely divided, it should be cut right across after a ligature has been applied on either side of the bleeding point. The object in view in this procedure is to allow of retraction and contraction of the severed ends of the vessel.

Treatment of Reactionary Hæmorrhage.—Unless due to the slipping of a ligature this amounts to nothing more than compression of oozing vascular points.

Treatment of Secondary Hæmorrhage.—This is a subject of sufficient importance to demand special notice.

1. If the hæmorrhage is but slight it may suffice to reopen the wound, remove the blood coagulum, wash out with a hot antiseptic solution, and apply a compress or stuff the wound with double cyanide gauze.

2. Should the bleeding be severe, a tourniquet must be applied, or the main artery compressed digitally. The wound must be reopened and the bleeding vessel sought for and ligated. Should this be impossible owing to the softened, sloughy state of the vessel walls, the main artery must be ligated on the proximal side of the bleeding point by a fresh incision through healthy tissues.

3. Should this method fail, the artery may be ligated still higher up, or in the case of an extremity the limb may be amputated.

4. In cases of even slight secondary hæmorrhage it is a wise precaution to have, in the case of a limb, a tourniquet loosely applied above the wound, so that it may, in case of a sudden gush of blood, be readily tightened by the nurse in attendance. In places where a ligature on the proximal side of the bleeding point cannot be applied, the actual cautery may be applied and the wound packed firmly with antiseptic gauze.

Hæmorrhagic Diathesis. *See* Hæmophilia.

Hæmorrhoidal.—This term is applied to the arteries, veins, and nerves which are distributed to the rectum and its neighbourhood (*e.g.* the *hæmorrhoidal plexus*); it also means "relating to hæmorrhoids" (*q.v.*).

Hæmorrhoids. *See* Rectum, Diseases of (*Hæmorrhoids*); *see also* Gout (*Irregular, Circulatory System*); Heart, Myocardium and Endocardium (*Effects of Cardiac Disease, Passive Congestion of Gastro-Intestinal Tract*); Liver, Diseases of (*Functional, Symptoms*); Malingering (*Digestive System, Simulation of Hæmorrhoids*); Nephritis (*Chronic, Renal Cirrhosis, Symptoms*); Pregnancy, Affections and Complications (*Cardio-Vascular Disturbances*); Prostate Gland (*Hypertrophy, Symptoms*); Stomach and Duodenum (*Morbid Anatomy, Gastric Hæmorrhoids*); X-Rays (*Therapeutic Applications*).

Hæmoscheocele.—Distension of the scrotum with effused blood.

Hæmosialemesis.—Vomiting of blood-stained saliva.

Hæmosiderin.—An iron-containing pigment derived from the decomposition of hæmoglobin in the tissues; it is said to be formed on the outside of the mass of extravasated blood, and so to differ from hæmatoidin, which is formed in the inside of the mass.

Hæmosporidia. *See* Parasites (*Protozoa, Hæmosporidia*).

Hæmostasis.—The arrest of hæmorrhage.

Hæmostatics.—Drugs or means for stopping bleeding; styptics; astringents; many of them also check discharges (*e.g.* from the intestine in diarrhœa and from the vagina in leucorrhœa). *See* Astringents; Copper; Ergot; Ferrum; Hamamelis; etc. etc.

Hæmotachometer.—An instrument for estimating the velocity of the blood current (Vierordt).

Hæmothorax.—The presence of effused blood within the thoracic cavity. *See* Aspirator, Uses of (*Therapeutic Purposes*); Chest, Injuries of (*Viscera, Wounds of*); Pleura, Diseases of (*Hæmothorax*); Pleura, Surgical Affections of (*Injuries, Hæmothorax*).

Haffkine's Prophylactic. *See* Cholera, Epidemic (*Prophylaxis*); Plague (*Curative and Preventive Sera and Prophylactics*).

Hair. *See* Skin, Anatomy and Physiology (*Epidermal Appendages*); Skin, Pigmentary Affections of (*Pigmentary Anomalies of Hair*); *see also* Alopecia; Electrolysis (*Use as a Depilatory*); Fœtus and Ovum, Development of (*Lanugo*); Ovaries, Diseases of (*Dermoid Cysts*); Physiology, Excretion by the Skin (*Hair*); Sclerodermia (*Circumscribed, Morphœa*); Skin, Parasites (*Examination of Hair*); Stomach, Surgical Affections (*Foreign Bodies, Hair-balls*); Syphilis (*Secondary, Affections of Hair and Nails*); Thyroid Gland, Medical (*Exophthalmic Goitre, Clinical Features, Hair*); Toxicology (*Lead Poisoning from Hair Dyes*).

Hair, Downy. *See* Lanugo.

Hair Tumours. *See* Stomach, Surgical Affections (*Hair-balls*).

Hairy Heart.—When the heart is covered with long shreds of fibrin (as in acute fibrinous pericarditis) it assumes a peculiar shaggy appearance—cor villosum.

Haldane's Hæmoglobinometer. *See* HÆMOGLOBINOMETER.

Halitus.—A vapour, odour, or exhalation, e.g. *Halitus oris fœtidus*, a stinking breath.

Hallopeau's Dermatitis. *See* DERMATITIS HERPETIFORMIS (*Synonyms*, *Types*).

Hallucinations. *See* ALCOHOLISM (*Chronic*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Hyperæmia*, *Symptoms*); DELIRIUM TREMENS (*Symptoms*); HYPNOTISM; PARANOIA.

Hallux.—The great toe, e.g. *hallux rigidus* or *h. malleus*, the condition in which the first phalanx of the toe is fixed at an angle of from 30° to 60°, while the second phalanx is extended; *hallux valgus*, in which the great toe deviates outwards (*i.e.* away from the middle line of the body), and leads to the formation of a bunion; and *hallux varus*, or pigeon-toe, in which it deviates inwards, and so comes to be separated from the other toes. *See* DEFORMITIES (*Foot and Toes*).

Halogens. — The elements chlorine, bromine, and iodine are called halogens, because by their union with metals they form bodies (salts) resembling ordinary salt (chloride of sodium); and the salts thus produced are sometimes called *halloid* (*i.e.* like sea salt). *See* CHLORINE; BROMUM; IODINE.

Halsted's Operation. *See* HERNIA (*Radical Cure, Inguinal*).

Halteridium. *See* MALARIA (*History*); PARASITES (*Protozoa*, *Hæmosporidia*).

Halys. *See* SNAKE-BITES (*Viperine Snakes*, *Crotalidæ*).

Hamamelis.—Both the dried bark and the leaves of *Hamamelis virginiana*, or Witch-hazel (United States), are official. The important constituent is *Tannic acid*, of which the bark contains more than the leaves. *Preparations*—1. Tinctura Hamamelidis. *Dose*—$\frac{1}{2}$-1ʒ. 2. Extractum Hamamelidis Liquidum. *Dose*—5-15 m. 3. Liquor Hamamelidis. 4. Unguentum Hamamelidis. 5. Hazeline (N.O.), a distilled extract from the leaves (represented in the U.S.P. by Aqua Hamamelidis), is probably the most efficient preparation of all. *Dose*—$\frac{1}{2}$-2ʒ. "Pond's Extract," a concentrated variety, is a popular American preparation.

Hamamelis is used locally as an astringent and hæmostatic in sore throat, pharyngitis, coryza, epistaxis, piles, and in bleeding from tooth-sockets, gums, etc. For these purposes it is diluted with water to the extent of 20-60 m. of hazeline, or of the tincture, to the ounce of water. Solutions of similar strength may be injected into the bladder in vesical hæmorrhage. The ointment is used in the treatment of hæmorrhoids. Given internally Hamamelis may check a simple diarrhœa. It is also said to act as a remote hæmostatic, but this is very doubtful. Good results, however, have been claimed for it in hæmoptysis, hæmatemesis, uterine oozing, and bleeding from the bladder.

Hammam Melouan. *See* BALNEOLOGY (*Africa*, *Algiers*); MINERAL WATERS (*Earthy and Calcareous*).

Hammerman's Cramp.—Smith's palsy. *See* NEUROSES, OCCUPATION.

Hammer Toe.—A deformity, usually of the second toe, in which the first phalanx is dorsiflexed, the second shows plantar flexion, and the third is extended. *See* DEFORMITIES (*Hammer Toe*).

Hamstring Jerk. *See* SPINAL CORD, MEDICAL (*General Symptomatology*, *Reflexes*).

Hamulus.—A hoop-shaped structure, *e.g.* the hamulus pterygoideus or hamular process of the pterygoid bone.

Hand. *See also* AMPUTATIONS (*Upper Extremity, Hand, Wrist Joint*); ARTERIES, INJURIES OF (*Wound of Palm of Hand*); ARTIFICIAL LIMBS (*Hand*); ASEPTIC TREATMENT (*Contact Infection and Disinfection of Hands*); DEFORMITIES (*Hand and Fingers*); ECZEMA (*Hands*); FINGERS; INFANT FEEDING (*Hand-Feeding*); LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies, Hand Presentation*); LABOUR, OPERATIONS (*Version*); MENTAL DEFICIENCY (*Cretinoid Cases, Character of Hands*); PHYSIOGNOMY AND EXPRESSION (*Movements*); PUERPERIUM, PATHOLOGY (*Infection, Causes, Prophylaxis*); TERATOLOGY (*Malformations of Limbs*); TETANY (*Motor Symptoms, Spasms of Hands*); TOXICOLOGY (*Chronic Arsenical Poisoning*).

SURGICAL ANATOMY.—The extensor tendons and superficial veins are seen on the dorsum. Situated respectively on the outer and inner sides of the palm are the thenar and hypothenar eminences, the former being composed of muscles acting on the thumb, the latter consisting of the small muscles of the little finger. The skin of the palm is thick, and contains neither hair nor sebaceous glands, but possesses an unusual number of sweat glands. The subcutaneous tissue in the palm is sparse, but the skin is well supported by the palmar fascia, and thus enabled easily to resist the pressure to which it is so frequently exposed. This fascia, too, affords protection to the underlying vessels and nerves. The superficial palmar arch crosses the palm in a manner represented by a curved

line running from the pisiform bone, and the lowest part of the arch being in the middle of the palm at a level of the web between the thumb and index finger. The deep palmar arch lies a quarter of an inch higher up the palm.

Appearances in Certain Diseases. — In examining the hands one should notice whether there be any cyanosis or œdema, the latter being most noticeable on the dorsum. The strength of the grasp should be ascertained, and, if deficient, recorded by the dynamometer.

The *shape* of the hands may be abnormal in consequence of some general disease. Thus in *acromegaly* the hands, though not deformed, are much enlarged and extremely broad, the enlargement being mainly due to increase of the soft parts. The palmar creases are unusually distinct. In *pulmonary osteoarthropathy* the hands are enlarged, but also somewhat deformed, and the fingers are clubbed at their extremities. In *syringomyelia* the hands may be enlarged, but they too show deformity, for instance, muscular atrophy, contraction, and scarring. In *myxœdema* the dorsum is swollen, but does not pit on pressure and the hands are cold. The hands of a *cretin* are short, broad, and appear somewhat swollen, are sometimes of a purplish colour, and the skin is crumpled, redundant, and cold. In *achondroplasia* the hands somewhat resemble those of a cretin, but the ring and middle fingers are curved away from one another, and the skin is more natural than in the cretin. The metacarpal portion of the hand in *Mongolian imbecility* is small and soft, while the fingers, as compared with the hand, are somewhat thick, but taper at their extremities, and the little finger has often a curve, the concavity of which is towards the ring finger.

General atrophy of the hand is chiefly observed as a congenital defect or as a result of infantile paralysis. Wasting of the soft parts is seen in the course of general wasting diseases, starvation, and old age, whilst atrophy, localised to certain areas, indicates some myelopathic or nervous lesion. Thus one sees atrophy of the thenar and hypothenar eminences in progressive muscular atrophy, and sometimes in syringomyelia and lead palsy. Localised atrophy may be associated with *contraction* and *deformity*, as occurs in ulnar paralysis from wasting of the interossei and the two inner lumbricales, the contraction affecting specially the little and ring fingers of one hand, or again in the *main en griffe* of progressive muscular atrophy, where all four fingers are equally involved.

The alteration in form may exist chiefly in the *joints*, which may be swollen, painful, distorted, etc. The causal condition will in most cases be rheumatism, rheumatoid arthritis, or gout, and in gouty subjects one may also find tophi, Heberden's nodes, and exostoses. In consequence of disease or injury of a peripheral nerve the *nutrition of the skin* may be impaired in the area supplied by it, and the skin becomes thin and shiny, or is even the seat of other trophic lesions.

Fibrillary twitchings in the muscles are seen in several conditions, *e.g.* progressive muscular atrophy, amyotrophic lateral sclerosis or syringomyelia, but the first-mentioned of these conditions is the most frequent cause. As regards *tremors*, the most important, from a clinical standpoint, is the tremor so often seen in the hands in chronic alcoholism.

Wounds.—The hand is one of the regions of the body most frequently wounded, and the majority of the wounds are on the palmar aspect. A small punctured wound of the palm may be troublesome on account of hæmorrhage from a punctured vessel. This is best dealt with by enlarging the wound and applying a double ligature to the injured vessel. Fragments of glass, broken needles, and other *foreign bodies* often penetrate, and chiefly into the palmar surface. They may be followed by suppuration, or may only give rise to pain when the skin over them is touched. It is often difficult to locate with accuracy, and still more difficult to remove these foreign bodies.

Septic Inflammation may occur after a wound of the hand itself, or may spread to the hand from elsewhere. Thus it is common after a whitlow, and more especially after a whitlow of the thumb or little finger, in which digits the flexor sheaths continue upwards through the hand to above the anterior annular ligament. The symptoms are in general the same as those of suppuration elsewhere, but in the palm, owing to the density of the more superficial tissues, any deeply-seated inflammation is liable to be unusually painful, and if pus be formed, it tends to travel either above the wrist towards the fingers, or towards the dorsum of the hand.

Tenosynovitis, or inflammation of the tendon sheaths, may be seen affecting the flexor or extensor sheaths in the hand or the fingers. If it be acute, there is pain, stiffness, creaking on movement, and sometimes fluctuation. Or the acute form may be of a suppurative character, and then tends to cause necrosis of the tendon and suppuration round the sheath. Pus in the palm travels to the regions previously indicated. Chronic tenosynovitis is usually tubercular, and occurs more often about the wrist than in the hand itself or the fingers. The affected sheath contains a variable amount of fluid in which one finds the characteristic melon seed bodies.

Compound palmar ganglion, which is usually a tubercular tenosynovitis of the common flexor sheath, is characterised by a chronic swelling in the palm and in the lower part of the forearm. This swelling appears to be divided by the anterior annular ligament into two portions, but the fluid can be driven from one portion to the other beneath the ligament. Simple ganglion, too, may be seen in the palm, but

the condition is dealt with elsewhere. (See Ganglion.)

The Bones of the hand may be affected by any of the ordinary bone diseases, such as periostitis, osteomyelitis, necrosis, or tuberculosis. Mention may, however, be made of *dactylitis*, which mainly occurs in the form of tuberculous disease of the phalanges in children, less often as a gummatous periostitis in syphilitic patients. The differential diagnosis of these two forms of dactylitis rarely presents much difficulty.

Tumours of the hand. The most important are the chondromata, cartilaginous tumours of benign nature. They are firm, rounded, often multiple, and may produce great deformity and uselessness of the hand.

Handwriting. *See* General Paralysis (*Symptoms, Handwriting*); Insanity, Nature and Symptoms; Mirror-Writing.

Hanging. *See* Asphyxia (*Causes*); Medicine, Forensic (*Death from Hanging*).

Hangnail.—A hangnail or agnail is a piece of epidermis attached by one end to a point near the border of the nail.

Hannay's Method.—A plan of preventing plumbism among white lead workers; "sulphate of lead is prepared from the sulphide, which is volatilised and oxidised in a furnace, and the vapours are passed through a spray condenser." (Lewis and Balfour.)

Hanot's Disease. — Hypertrophic hepatic cirrhosis with chronic jaundice. *See* Liver, Diseases of (*Biliary Cirrhosis, Hypertrophic*).

Hansen's Bacillus. *See* Leprosy (*Pathology of Lepra Tuberosa*).

Hapalonychia.—Softness of the nails due to defective cornification (Gr. ἁπαλός, tender, and ὄνυξ, a nail).

Haphalgesia.—A sensory condition in which a simple touch causes intense pain (Gr. ἁφή, touching, and ἄλγησις, sense of pain). *See* Hysteria (*Sensory Disorders, Dysæsthesia*).

Haphephobia. — The morbid fear or dread of being touched.

Haplo-.—In compound words *haplo-* (Gr. ἁπλόος, onefold or single) means simple, *e.g.* *haploacne*, acne simple, *haplomelasma*, simple melasma, *haplophyma*, a simple tumour, and *haplotomia*, a simple incision.

Haptins.—Receptors. *See* Immunity.

Haptophore Group.—The combining portion (Gr. ἅπτω, I seize) of a toxin, that part which unites in constant proportions with the antitoxin; the other portion, which exerts the poisonous action, is the *toxophore group* (*Ehrlich*). *See* Immunity.

Hardness of Water. *See* Water (*Hard, Methods of Softening*).

Hard Soap.—Sapo durus; sodium oleate, containing 30 per cent of water. *See* Sapo.

Hare-Lip. *See* Palate (*Malformations of Mouth, Hare-Lip*); *see also* Cheek, Fissure of; Hydrocephalus (*Complications*); Mental Deficiency (*Abnormalities of Physical Formation*).

Harlequin Fœtus. — The peculiar appearances produced by fœtal ichthyosis have suggested this name. *See* Ichthyosis.

Harpoon. — An instrument used for obtaining a fragment of a deep-seated tissue for microscopic examination.

Harrison's Groove.—The transverse groove on the chest wall passing outwards from the ensiform cartilage toward the axilla, seen in rickets; it is seen also in cases of chronic tonsillitis.

Harrogate. *See* Balneology (*Great Britain, Sulphur Waters*); Mineral Waters (*Chalybeate, Muriated Sulphur*); Therapeutics, Health Resorts (*English*).

Hartley-Krause Operation. *See* Nerves, Neuralgia (*Trigeminal, Treatment, Removal of Gasserian Ganglion*).

Harvest Bug. *See* Scabies (*Other Acari, Leptus Autumnalis*); Stinging Insects (*Leptus Autumnalis*).

Harvey's Discovery. *See* Medicine, History of (*Seventeenth Century*).

Harz Mountains. *See* Therapeutics, Health Resorts (*Germany*).

Haschisch or **Hasheesh.** *See* Cannabis Indica; Toxicology (*Indian Hemp*).

Hassall's Corpuscles. *See* Physiology, Internal Secretions (*Thymus*); Thymus Gland (*Anatomy*).

Hastings. *See* Therapeutics, Health Resorts (*English*).

Haustus. — A draught; a liquid medicinal preparation to be taken as a single dose. *See* Prescribing.

Haut Mal.—The fully-developed form of epilepsy; epilepsia gravior. *See* Epilepsy.

Haversian Canals, Spaces, and System. *See* Physiology, Tissues (*Bone*).

Hay Asthma. *See* Asthma (*Derivation*); Hay Fever.

Hay Fever.

See also Adrenal Glands, Adrenalin (*Uses*); Asthma (*Derivation*); Cocaine (*Uses*).

The study of hay fever is a matter of recent years, the first good account of it being written by a Dr. Blackley of Manchester in 1873. He was a great sufferer himself, and wrote an admirable brochure on it. He was the first to substantiate the point that the commonest source of irritation is the pollen of certain grasses, especially of ripe rye-grass. He also noticed the fact that it was only the few who experienced any discomfort from it, though all were equally exposed to it. The next writer on the subject was Dr. Beard of New York, who, five years after, pointed out that nearly all the patients were of the neurotic temperament, and that constitutional treatment directed towards the amelioration of this condition was often of considerable value. Then Dr. Marsh, also of New York, in 1877 published a paper on hay fever. He himself was susceptible only to the pollen of the common ragweed (artemisia), which blossoms only in the autumn in America. But he was obviously mistaken in supposing such patients were susceptible to this alone, as in that case the hay fever of those suffering in the spring would be unaccounted for. In the year 1882 Dr. Daly of Pittsburg contributed perhaps the most important theories of all, though he in his turn was practically misled. He maintained that hay fever was invariably due to morbid conditions of the nose, and that they could all be successfully treated by intra-nasal operations. But cases are frequently encountered in which the nose is absolutely normal, and such would be inexplicable on this supposition. Sajous in the following year made a contribution of some value, in which he corroborated Daly's theories, which were subsequently embraced by specialists all over the world—in Vienna by Hack, and in this country by Woakes, M'Bride, and many others. But Dr. J. N. Mackenzie of Baltimore advanced the theory that the coryza, as he considers it, is dependent upon some functional derangement of the nerve-centres rather than any abnormality in the mucous membrane. Then there appeared Morell Mackenzie's able treatise in 1885, in which he contended that there was no intra-nasal disease at all, and that the whole trouble was an idiosyncrasy dependent upon the general health. All these writers were practically right; and yet not one of them, as appears to me, has grasped the subject from a wide point of view. I think we may define hay fever, in the first place, as an idiosyncrasy which renders certain individuals abnormally sensitive to sources of irritation to which they, in common with the rest of their fellow-creatures, are exposed; which idiosyncrasy may be augmented, if not actually started, by abnormal conditions of the nose. Yet it must be admitted that we find many a case in which the mucous membrane is exquisitely sensitive to these unwarrantable sources of irritation, and, nevertheless, in which we find no morbid condition whatever in the nose. Yet, again, although there may be abnormalities in the nose, they do not justify our saying that the hay fever is actually caused by such abnormal conditions. Rather may we assert that when the affection has persisted for many years it may actually lead to pathological conditions.

Now I must enlarge a little further on these points. We may have abnormalities in the nose interfering with respiration, yet in themselves not necessarily pathological, but merely malformations. We may have many forms of obstruction to respiration from hypertrophy, such as spurs and deviations of the septum. These are common causes of obstruction, and when they occur to any extent they more frequently obstruct the inferior meatus. And it seems to me, rather from clinical observation than from any theoretical consideration, that it is essential that these patients should breathe easily through the inferior meatus. In all patients there is a strong instinct to breathe through the nose rather than through the mouth, often in spite of grave obstruction, though perhaps the instinct does not assert itself so forcibly in adults as in children. When the inferior meatus is obstructed, the inspired current of air, instead of passing along the less sensitive portions of the nose, is drawn up into the middle meatus, which is more sensitive and irritable than the lower. So that in many cases of hay fever the history is this: at the starting-point we have the idiosyncratic hyperæsthesia, the consequence of which becomes more serious if the inferior meatus be obstructed; next, as the consequence of the continued irritation, actual hyperplasia of the inferior turbinated bodies is induced. Yet we must admit on clinical grounds that these cases of real hyperplasiæ offer as good a prognosis for surgical treatment as those in which the obstruction is structural rather than pathological.

As to the conditions under which the affection may begin, we have predisposing causes as well as those immediately exciting. In the first place there is *climate*; hay fever occurs in temperate latitudes, being in tropical countries almost unknown. Then we find that the idiosyncrasy is confined to certain *races*, being especially found among the Anglo-Saxons and the associated portions of the Celtic race: the Irish are certainly not exempt. *Heredity* is a factor of the greatest importance. I have in my books the case of a mother and two

daughters who suffer from hay fever; a third daughter suffers from paroxysmal sneezing all the year round, although she does not have hay fever; one son suffers severely from hay fever, and another from nasal asthma—that is to say, bronchial spasmodic asthma cured by intra-nasal treatment. All these cases presented abnormal conditions of the nose. Sajous maintains that in 35 per cent of all his cases there was well-marked evidence of heredity, and that 42 per cent had asthmatic relatives. Next we find also that *class* has much to do with the tendency to sneeze—the educated classes being much more prone to the affection than the uneducated. Mackenzie said he never had a case among his hospital cases; but the hospital class is becoming better educated and more neurotic every day, and we certainly now find among them a fair proportion of hay fever cases. Then *town-dwellers* are more prone to it than country gentlemen, though I have seen several instances of it among the latter with apparently normal nerves and plenty of good blood. *Sex*, too, makes a difference, men, it is said, being more prone to the affection than women. Among other causes of predisposition, the long existence of catarrhal conditions renders patients more sensitive to those forms of irritation which occur in the summer. Of the direct sources of irritative attack of course the pollen of grasses, especially of rye-grass, is the most common, beginning to harass its victims about the first week in June, often much earlier, according to the degree of advance of the season. Some begin sneezing so early in spring that one is compelled to accuse the catkins; while the ragweed, which is confined to America, does not begin its attack until the autumn. Other patients are sensitive to nothing but roses. While the hay-fields are the misery of most patients, less commonly the moors and heather will make some men suffer when they go grouse-shooting. Others begin sneezing when exposed to the bright sunlight at sea, though the latter gives immunity to many who suffer from vegetable sources of irritation. There is a plant in Australia which induces sneezing; it flourishes in the spring, about September, and I believe it is commonly known as the cape-weed. It covers the hills around Adelaide to the height of some thousand feet or so. It is a composite, and the pollen is so profuse that after driving for two or three miles in the country the sides of the carriage will be covered with its yellow dust. I am told that most of the population of Adelaide are affected with hay fever during the time of the blossoming. Certain other people, again, only suffer from animal sources of irritation. One patient tells me she always sneezes when a cat comes into the room; and I know several who suffer from sneezing when in contact with horses.

The symptoms usually begin with a violent paroxysm of sneezing on waking early in the morning, sometimes supervening suddenly and sometimes more gradually. Occasionally, for many days before it actually begins, the patient will experience a feeling of irritation about the inner canthus of the eye, which he is compelled to rub continuously, or an itching about the alæ of the nose. In one of my patients the earliest symptom is a coldness and pallor of the nose, which, though it is warm sunny weather, he has to rub to restore the circulation. Sometimes the sneezing will last only for a few minutes, sometimes for many hours, and it is always accompanied by a profuse flow of watery mucus from the nose. A patient will sometimes assert that he saturates a couple of towels in the space of half an hour, after which he is naturally quite exhausted; indeed the collapse that sometimes follows is very distressing. Seldom are patients seen in greater misery than those suffering from hay fever—strong men become anæmic wrecks after six or eight weeks of the malady, which time is the ordinary duration, although many patients remain prostrate for as long a period after the symptoms have subsided. Of course if they go out of doors during these weeks the symptoms become aggravated, especially if there is wind and sun; so that they have to protect themselves in a very extraordinary way—with thick veils of gossamer over their faces, blue spectacles, and green umbrellas! Some of these patients suffer from more or less sneezing all the year round, these being especially cases where the continual attacks of hay fever have induced actual hypertrophy of the inferior turbinated bodies. Sometimes a patient is found who is sensitive to every source of irritation, not only to pollen in its various forms, but to emanations from animals. They suffer continually from dust; they cannot take a book from the bookshelves for fear of provoking sneezing; they dare not go near a feather bed or into a room after it has been dusted; and so on. The slightest changes of temperature will cause some swelling of their Schneiderian, adjacent surfaces will come into contact, and thus may the same train of symptoms arise even without the introduction of any irritating substances.

Usually the asthma of hay fever patients is a later symptom; the sneezing has persisted for ten days to three weeks before it begins, and sometimes the nose-symptoms abate when the asthma is fairly established. This probably occurs as soon as the swelling in the nose becomes so pronounced that nasal respiration is no longer possible, the bronchi themselves becoming thereby more exposed to the pollen, etc. Yet I must confess that many patients suffer from asthma alone without any sneezing symptoms at all; and it seems to me that these are more often cases of permanent obstruction to the nasal respiration.

A few words on prognosis must preface those in reference to treatment. In cases where we can find neither structural nor pathological abnormalities, the prognosis is bad. All we can hope to do for such patients is to mitigate their symptoms as much as possible. The cases that are best are those in which we find something demanding operation, and the bigger the obstruction the better the prognosis. The actual duration of the disease, whether the patient has suffered for few or many years, does not affect prognosis in any way. The most striking case of cure I have had was, without exception, the worst case in my experience. The patient, a man of intensely active mind and neurotic temperament, had for thirty years considered the whole of the summer a misery; yet he was completely cured by the removal of a large spur on the septum completely blocking the inferior meatus; and for ten years, so far as I know, he has had no return of symptoms. I do not think, either, that the general condition of the patient affects the prognosis; although it is of some consequence whether or no he be very neurotic. To differentiate a little further, the most suitable cases for surgical treatment are those in which we find the inferior meatus obstructed, whether by deviations or spurs of the septum, or by true hyperplasia of the erectile tissue; next to these come chronic engorgement of the inferior turbinated; next, adenoids in children. Less amenable are cases of polypus obstructing the middle meatus; and it is the fact of having seen polypi in typical hay fever cases, where the removal of the growths gave but little relief, which has inclined me to the belief that the restoration of breathing through an obstructed inferior meatus is the essential factor in treatment.

Further, we find cases in which there is no actual obstruction in either inferior or middle meatus. Opposite to the anterior extremity of the middle turbinal we often find on the septum curiously ill-defined œdematous and pallid swellings which obscure the view, and bring the septum into contact with the middle turbinal. Like engorgement of the inferior spongy bodies, they are boggy, and pit under pressure, while they collapse under cocaine in a few seconds. Destruction of these boggy swellings with the galvano-cautery is often attended with the happiest results. Presumably in these cases the trouble is due to direct irritation, which induces swelling of the mucous membrane where it abuts on the middle turbinal, thus evoking the whole train of symptoms. Such, on the whole, are good cases to treat. Let me repeat once more that the worst cases for treatment are those where we can find no structural or pathological abnormality to deal with. Where there is opportunity for operation we should advise it, although it is impossible to be absolutely sure of effecting a cure; indeed we seldom attain an *absolute* cure. Yet in this disease, as in all others of the nose, let us, in determining whether or no operation be necessary, remember M'Bride's dictum that we should judge the nasal fossæ by what they can do rather than by structural aberration from the strictly normal.

In cases where surgical treatment does not promise much, what can we do? The first and most common remedy is only mentioned as a warning against its use: it is cocaine. The mischief it induces is sometimes appalling, and the patients at the end of the summer are generally worse than if they had not used it at all. But the temporary relief it affords is unquestionable. Patients may begin with a 2 per cent solution, but are soon compelled to increase the strength rapidly, until they often end with a 30 per cent solution; and, finally, they become wrecks from the pernicious effect of the drug on the nervous system. I have seen two such cases in medical men, one of whom appeared to be developing symptoms of general paralysis, although he completely recovered as soon as he relinquished the habit.

But there are other local remedies of distinct help: the old-fashioned compound tincture of benzoin, the bismuth and morphia insufflations, known as Ferrier's snuff, both may give relief, and, on the whole, Ferrier's snuff is preferable to cocaine, although it does not arrest symptoms as decidedly. Sprays and lotions of borax, or boric acid, especially if combined with liq. hamamelis, are very soothing, and wash away accumulated secretion. The patient always likes such applications, though they must be used in very weak solution. A good method of application is for the patient to take the solution in a teaspoon, to hold the head back, and simply pour the warm fluid into the nose.

Another remedy I have occasionally found singularly useful has been a solution of chromic acid, $\frac{1}{16}$ or $\frac{1}{8}$ of a grain to the ounce, sprayed or poured into the nose three or four times a day or oftener, and held in the nose as long as possible. I had one patient who would carry about her spray of chromic acid just as others will carry about their cocaine. Another patient, after two or three weeks' use of it, seemed to be completely cured, and had no return of the symptoms the following summer. A good deal was talked a few years ago of using bichloride of mercury. Dr. Carl Genth used it in a $\frac{1}{3000}$ solution dropped into the eye, and allowing it to pass into the nose. I have heard two or three practitioners speak highly of it, and I have every reason to think it worth trying. Strong carbolic acid has been much vaunted as a cure; but I have seen so much mischief accrue from its incautious use that I cannot recommend it—at any rate so long as we have safer and more certain means of treatment. A 5 per cent solution of eucaine hydrochloride (A) some-

times gives great relief, though its application stings at first, and it does not act so strikingly as cocaine. But it does not exhibit the physiological disadvantages of the latter drug. Menthol sometimes relieves; and the following prescription I have found of the greatest service:—℞ Menthol gr. 5, eucaine (A) hydrochlor. gr. 5, zinci oxidi gr. 20, adipis lanæ ℥ss., paraff. liq. ad. ℥j. M. Ft. ung. Sig.: To be applied to the nostrils frequently with a camel-hair brush. During the last few months I have found suprarenal extract, 4 to 10 per cent, as a spray of signal use in a few obstinate cases of ordinary paroxysmal sneezing; and I suspect it will prove beneficial to some sufferers from hay fever.

Another point of importance in treatment is the careful feeding and resting of the patients, while they are probably all better for some stimulant. No special dietary is indicated, seeing that the patients present no tendency to lithæmia, etc. Various nerve tonics are also useful, like nux vomica, valerian, and asafœtida. Rather than give the patient cocaine, it might be wise to allow the opium pipe. Its use is risky, as patients grow addicted to it, and I have never had to prescribe it; but its power of controlling the worst symptoms is beyond all question.

[Dunbar's antitoxin, a serum produced by injecting the pollen of rye and other grasses into animals, has been favourably reported upon; it is applied to the eyes as the serum, and to the nasal mucous membrane as a snuff; it acts best as a prophylactic, and in established cases it modifies the severity of the symptoms.]

Haygarth's Nodosities.—Osteophytes growing on the knuckles in cases of arthritis deformans. *See* Rheumatism, Rheumatoid Arthritis.

"Hazeline."—A proprietary preparation of the bark of the twigs of witch hazel (*Hamamelis virginiana*); it is recommended in doses of one to three drachms in various hæmorrhages (hæmoptysis, hæmatemesis, etc.) and in diarrhœa and enteritis, and it is used externally in the form of Hazeline Cream and Hazeline Snow (B. W. and Co.) in eczema and other irritable skin affections. *See also* Hamamelis.

Head.

See also Alcoholism (*Acute*, *Diagnosis*, *Head Injuries*); Arteries, Ligature of (*Carotid*); Bandages (*Capelline*); Brain, Physiology; Brain, Surgery of (*Concussion*, *Compression*); Children, Clinical Examination of (*Head*); First Aid; Headache; Head-Shaking; Hydrocephalus; Labour, Physiology of (*Passenger*, *Fœtal Head*); Labour, Stages and Duration; Labour, Diagnosis and Mechanism; Labour, Management of; Labour, Prolonged, Faults in the Passenger; Labour, Operations (*Forceps*, *Embryotomy*); Lymphatic System (*Glands of Head*); Meningitis, Tuberculous; Meningitis, Epidemic Cerebro-Spinal; Physiognomy and Expression; Tetany (*Motor Symptoms*).

The *shape* of the head may be quite normal on examination, but there is often a definite abnormality produced by certain diseases.

In childhood a departure from the normal shape may be due to several causes. In well-marked *rickets* defective ossification is a prominent feature, the sagittal and coronal sutures remain open after the fourteenth month, the fontanelle does not close during the second year of life, and in consequence of defective ossification and thinning of the bones there may be areas which give on pressure and yield to the fingers a sensation of "parchment crackling." This condition is called cranio-tabes, and is usually most marked in the parieto-occipital region. If rickets lasts for some time there will be hyperostoses on the outer surface of the cranial bones, especially the frontals and the parietals, causing what are known as nodes or bosses, and the head becomes characteristically square. Neither the bosses nor cranio-tabes, however, are pathognomonic of rickets, as they are found in *congenital syphilis*, and are especially pronounced in those children who are the subjects of both these diseases. In congenital syphilis there is depression of the bridge of the nose, fissures about the mouth, Hutchinson's teeth, and other signs which do not occur in rickets. In *chronic hydrocephalus* the head is large and globular, the eyeballs are prominent from depression of the orbital plates, the superficial veins of the scalp are distended, the face is proportionately small, the head is with difficulty held upright, the sutures and fontanelles are widely open, and there is cranio-tabes. In *microcephaly* the head is small, the vertex tends to be conical, the forehead recedes very markedly, and the sutures and fontanelle close early. In *cretinism* the head is large as compared with the body, the forehead, as in microcephaly, is low and receding, but the anterior fontanelle remains open for years, and the eyes are wide set from one another. In *Mongolian imbecility* the head is short and spherical, and the eyes are set somewhat close to one another, while in *achondroplasia* the head is large and broad, the forehead is prominent, and the nose resembles that of a bulldog.

In *acromegaly* the cranium is often large, and the sagittal suture is palpable as a thickened ridge. The facial alterations are more pronounced, and consist chiefly in enlargement of the zygoma and frontal processes, and especially of the inferior maxillæ, whereby the face is much lengthened. Enlargement of the skull is also seen in *osteitis deformans*, but here the facial bones are hardly ever affected; and also

in those rare conditions known as *diffuse hyperostosis*, which affects the skull bones and occurs in youth; and, lastly, in *leontiasis ossea*, in which disease there are hyperostoses in the form of tumours growing from the skull bones, and especially the maxillæ, the disease commencing in childhood and producing great deformity in later life.

Symmetry.—The cranium of infants is often somewhat asymmetrical, either without definite cause or in consequence of rickets. Asymmetry of the head may be seen in *hemihypertrophy*, or specially affecting the face, as in *progressive facial hemiatrophy*, or more frequently in association with wry-neck, where there is imperfect development of the face on the same side as the affected sterno-mastoid.

In babies the anterior fontanelle normally closes between the fifteenth and eighteenth months. The conditions which retard closure have been already mentioned, *e.g.* rickets and chronic hydrocephalus. Examination of the fontanelle shows that its normal tension may be raised by crying or coughing, or by cranial tumours, meningitis, or hydrocephalus, which produce increased intracranial pressure, while the most common cause of depression of the fontanelle and lowering of its tension is severe and continued diarrhœa. The fontanelle has normally a slight pulsation communicated to it from the arteries forming the circle of Willis, but auscultation is useless in determining the presence of intracranial disease.

The percussion note of the skull is of doubtful value in diagnosis, but percussion is often helpful in locating such conditions as cerebral tumours and meningeal gummata, or in forming a diagnosis of mastoid inflammation.

Head retraction, or cervical opisthotonos, is an important sign in babies of meningitis, whether of tubercular or of the so-called simple form. In its mildest form there is merely rigidity of the neck, due to tonic spasm of the muscles at the back of the neck, but when well marked the head is fully extended, the occiput even touching the back, and in such a case the inflammation has probably spread to the spinal meninges. Head retraction is also seen in infants with chronic diarrhœa, or associated with cerebrospinal congestion, *e.g.* in typhoid fever. The condition must not be mistaken for wry-neck, or the stiff neck of rheumatism or spinal caries. In adults we find head retraction in tetanus and hystero-epilepsy.

Conjugate deviation of the head and eyes is due to cerebral hæmorrhage, meningitis, or tumours, but most often to hæmorrhage. The eyes and head are turned towards the side where the lesion exists, *i.e.* away from the paralysed side; but in the stage of early rigidity they may be turned in the opposite direction. In hæmorrhage of the pons the deviation is just contrary to that of other hæmorrhages.

Injuries of the Scalp; Scalp Wounds.—The edges of the wound do not gape if the cranial aponeurosis be uninjured, and the appearance of a wound inflicted with a blunt instrument will closely resemble that of an incised wound. If the aponeurosis has been divided there is much gaping, and the scalp can be freely stripped off from the pericranium. Scalp wounds usually give rise to severe hæmorrhage, because the vessels which lie in the fibrous tissue of the scalp are unable to retract. The wounds heal readily. The chief dangers are hæmorrhage and suppuration in the space between the pericranium and the aponeurosis of the occipito-frontalis. If the pericranium has been detached from the bone by violence or suppuration there is merely slight superficial necrosis of the bone, which is hardly in any way dependent on the pericranium for its blood-supply. It is of extreme importance to ascertain whether there is a fracture of the skull in addition to the scalp wound. Thorough investigation of the wound is imperative, and if in doubt it is a good rule to enlarge the wound of the soft parts, and explore with the aseptic finger, so as to avoid mistaking torn pericranium for a fracture, as one may do if a probe be used. Of less importance as local signs of fracture are (1) an infiltration of blood into the conjunctiva and eyelids, most commonly seen in fractures involving the orbital plate or the orbital ridge, and which must not be mistaken for the infiltration following a local injury to the eyelids; and (2) the escape of blood or cerebro-spinal fluid from the ears, mouth, or nose after fracture of the base.

Cephalhæmatoma is a blood tumour of the scalp caused by trauma. According to its position in the scalp we speak of it as (1) subcutaneous, (2) subaponeurotic, or (3) subperiosteal. The hæmatoma may form a well-defined swelling, which, with a soft and somewhat depressed centre and a firm, elevated margin, may resemble a depressed fracture. The diagnosis of hæmatoma is determined by exerting pressure on the elevated margin, which is then found to disappear.

Subaponeurotic hæmatoma is not common, but if it occur the blood is only limited by the attachments of the occipito-frontalis. The subperiosteal variety is confined by the pericranium to one bone, and is most often seen in infants over the right parietal bone after forceps delivery.

The treatment of scalp wounds requires no special mention, further than emphasising the importance of thorough cleansing and shaving of the scalp for a considerable area around the wound.

Cephalhæmatomata are best left alone, for the blood will be absorbed. Strapping, pressure, evacuation of the blood, or incision and drainage on account of suppuration, are rarely required.

Tumours of the head are mostly seen in the scalp. They may be simple sebaceous cysts, or

dermoid cysts. The latter are most common about the outer angle of the orbit, yet may be found in the middle line, and then closely resemble meningocele. Epithelioma, fibroma, the various forms of angioma, and more particularly the so-called cirsoid aneurysm, and sarcoma may be mentioned. By cephalhæmatocele is meant a cephalhæmatoma, which communicates with one of the sinuses of the dura mater, causing a small reducible swelling, which may pulsate. Lastly, there are the congenital malformations of the skull, causing cephalocele, the most common variety being the meningocele, which, as a rule, occurs in the middle line, and is more or less reducible and translucent.

Diseases of the Bones.—Osteomyelitis occurs as a result of compound fracture (*q.v.*). Tuberculosis of the cranial bones is a very chronic condition, in the course of which a tubercular abscess forms. This bursts, and sinuses remain running through the bone.

Syphilis gives rise to gummata in the pericranium or bone, or between the bone and dura mater. They either disappear under treatment, burst externally, or become ossified, and then form "syphilitic exostoses." The head in congenital syphilis and in other general diseases has been already referred to.

Headache.

See also Bile and Biliousness (*Bilious Headache*); Brain, Affections of Blood-Vessels (*Anæmia, Syphilitic Vascular Lesions*); Brain, Tumours of; Brain, Inflammations; Brain, Surgery of; Brain, Cerebellum, Affections of (*Hæmorrhage*); Climacteric Insanity; Gout; Meningitis, Tuberculous; Meningitis, Epidemic Cerebro-Spinal; Nephritis (*Chronic, Renal Cirrhosis*); Ocular Muscles, Affections of (*Paralysis, Diagnosis*); Refraction (*Refractive Headaches*); Rheumatism in Children (*Symptoms, Nervous Affections*); Stomach and Duodenum, Diseases of (*General Symptomatology*); Typhoid Fever (*Symptoms*); Uræmia (*Chronic Symptoms*); etc. etc.

Headache has been aptly described by Handfield Jones as a *Dis-ease.* It is a symptom of a great variety of morbid conditions, and frequently the discovery of its cause is a problem of great difficulty. In all cases the pain is perceived in the sensorium. The seat of its production may be: (*a*) nerve-endings, in the meninges, the scalp, some organ of special sense, or, at a greater distance, in the stomach, or some other organ; (*b*) irritation at any part in the course of a nerve; (*c*) or it may arise from the condition of the nerve-cells of the sensorium itself, or of the blood surrounding them.

The causes of headache have been divided into *organic* and *functional.* The investigations of Hodge on the changes produced by fatigue in the nerve-cells of the bee render it probable that, with improved methods and more exact knowledge, the class of functional disorders, with the exception, perhaps, of those due to reflex irritation, will disappear from our nomenclature. As a factor in the production of headache, the quality of the sentient mechanism of the individual plays a most important part; some persons inform us that they never had a headache in their life; in certain individuals a small elevation of temperature is always accompanied by pain in the head, whereas high temperature causes no headache to others. Women are more liable to this kind of suffering than men; it is more frequent in persons of nervous temperament than in others. Age brings often a general blunting of the sensibility both of nerve-centres and of end-organs, and headache, as a rule, recurs with diminishing frequency and severity as life advances. Occasionally more than one cause operates in the same case; *e.g.* in fevers both hyperæmia and toxæmia act.

The causes of headache may be arranged in the following order:—

1. *Intracranial Disease.*—Acute and chronic inflammation of the meninges from syphilis, gout, rheumatism, tubercle, chronic alcoholism, chronic kidney disease, general paralysis, and other forms of insanity. Head injuries occasionally produce headache for a long time after the injury, although there may not have been external mark. Encephalitis, hydrocephalus, intracranial hæmorrhage, abscess, tumour. Small hæmorrhages and slowly-growing tumours in the substance of the brain are not accompanied by pain. Bramwell points out that tumours cause pain by (*a*) increase of intracranial pressure; (*b*) direct involvement of the membranes; (*c*) direct implication of the fifth nerve.

2. *Cranial and Pericranial Affections.*—Any disease which produces pressure on nerve-trunks or twigs as they pass through foramina in bone or membrane—syphilitic, rheumatic, and malignant diseases of bone or of pericranium,—erysipelas, wens, excessive weight of hair, certain styles of coiffure, pediculi, tight or too heavy head-dress.

3. *Reflex Irritation.*—In addition to some causes named in class 1, and most in class 2, there are those from the organs of special sense and the abdominal organs. Irritation of the end-organs of special sense is a powerful exciting cause of headache because of the extreme sensitiveness of these structures.

(*a*) *Nose.*—Any disease of the nose and of its accessory cavities. Dundas Grant says that adenoids in the naso-pharynx are the most common form of nasal disease producing headache. Acute congestion of frontal sinuses in

colds and in influenza cause severe frontal headache. Treatment of chronic disease of frontal, antral, ethmoid, and sphenoid sinuses has been followed by cure of chronic pain in head.

(*b*) *Eye.*—Muscular and retinal strain in a healthy eye, as by a visit to a picture-gallery; also the effect on the retina of glare from the sea, sand, snow, bright roads. Chronic or frequently-recurring headache may be induced by organic diseases of the eye, such as glaucoma and iritis. In chronic headache the ocular tension should always be carefully tested. Errors in the optic apparatus which induce fatigue are very frequent causes of headache:—1. These may affect the focussing: (*a*) errors of refraction—hypermetropia and astigmatism, less frequently myopia and anismetropia; (*b*) weakness of the ciliary muscle—following diphtheria, due to presbyopia or to debility. 2. Errors in powers of fixation—esophoria, exophoria, hyperphoria—due to an excess or to a deficiency in the action of the muscles acting on the globe.

(*c*) *Teeth.*—The eruption of teeth both in first and second dentition; also of the wisdom teeth. This is a more probable cause when the jaw is small and the teeth impacted. Caries specially of teeth in the upper jaw.

(*d*) *Ear.*—Wax in the meatus, otitis media, cholesteatoma in the mastoid antrum. Legal points out that sometimes ear disease causes not so much pain in the ear as in some other part of the head.

(*e*) *Digestive tract.*—Occasionally we find that headache comes on after, or is aggravated by, the taking of food, and persists till the stomach is empty. Dilatation of stomach causes headache, probably through the production of ptomaines. Overloaded bowels may act in the same way, but overloaded bowels and rectum and hæmorrhoids also act by reflex irritation. Lumbricoids and tape-worm should also be borne in memory as causes in obscure cases.

(*f*) *Reproductive system.*—Although Matthews Duncan stated that the generative organs do not cause pain above the breasts, it is established that congestions and displacements of the uterus do occasionally give rise to headache. That which is frequent in young women about puberty and in older women at the climacteric is more directly related to the condition of the nervous system.

4. *Toxæmia.*—(*a*) Autogenetic, from uræmia, liver diseases, gout, rheumatism, oxaluria, constipation; (*b*) Heterogenetic, from alcohol, impure air of crowded meetings, badly-ventilated bedroom, bad drainage, certain drugs, such as opium, quinine, etc., malaria, fevers. It is to be borne in memory that the liver, the largest gland in the body, stands at the gateway between digestion and circulation, and in deranged conditions is liable to permit the passage of ptomaines. It is often deranged without prominent digestive symptoms.

5. *Hyperæmia.*—Acute congestion in encephalitis, plethora, menstrual suppression, increased action of heart, violent exertion, and mental excitement. Passive congestion from certain diseases of the circulation and pressure of tumours on great veins leading from the head.

6. *Anæmia and Nervous Debility.*—Chlorosis and the various diseases which produce general anæmia induce debility of nerve-centres, and in some constitutions headache results. Overwork, prolonged lactation, exhaustion, mental anxiety—which Laycock called phrenalgia: he considered depression of spirits and hypochondriasis to be varieties of phrenalgia—neurasthenia, hysteria, sunstroke.

7. *Migraine* or *megrim.*—This is a paroxysmal form of headache allied to epilepsy and other nerve-storm diseases. As it differs materially from other forms of headache, it requires notice at greater length. It is hereditary, occurs in families of nervous constitution, in those who are intellectual and studious, and in members of families with a history of epilepsy, chorea, insanity, and nerve weakness. Although there is a case on record which commenced at the early age of three and a half years, it most often begins at puberty, diminishes in frequency and severity with advancing age; it generally ceases in old age, but not invariably so. About the menopause it generally becomes temporarily aggravated. The attacks are periodical—once a week or at longer intervals; in women it frequently occurs at the menstrual period, commonly just before it. An attack may last a few hours or several days, most frequently one day. Its onset is occasionally indicated by prodromata—in some persons a feeling of well-being, in others malaise, or sleepiness or constipation; frequently there are optical phenomena—dimness or confusion of vision, coloured lights, flashing, dazzling, hemiopsia, or teichopsia. The attack generally comes on in the early morning, either immediately on awaking or soon after, with pain or throbbing in one or both temples; there may be pallor or flushing of the face, or both may occur at the same time. The pulse is slow in proportion to the severity of the pain, the body is cold, the head hot. In severe cases the subject must rest—generally lying, but some must sit—in a darkened room; the pain is continuous with occasional exacerbations, which may be agonising. After a time nausea and vomiting occur, and following this the subsidence of the attack; in others vomiting recurs and is bilious; rarely diarrhœa is a symptom. During the attack the urine and saliva are diminished, and usually there is anorexia. Haig states that during the prodromal period of well-being the excretion of uric acid is below the normal, and that during the seizure it rises above it. Many theories have been advanced as to the cause of migraine. Haig believes that uric-acidæmia is to blame; some consider that

the primary error lies in the digestive system, and in order to cure an attack and prevent recurrence treat the stomach; others locate the error in the circulation; others in the nervous mechanism. Uric-acidæmia does not produce migraine in every subject, only in those with a peculiar nervous system. The circulatory phenomena, the headache, sickness, uric-acidæmia, are not the disease, but incidents in the attack. In migraine, as in other nerve-storm diseases such as epilepsy, there are probably at least three factors. 1. There must be a peculiar constitutional condition of the nerve-centres. 2. A morbid or toxic condition of the blood. 3. Some peripheral irritation. This irritation acts as the exciting cause; it may be worry, excitement, overwork, powerful impressions on any of the special senses, errors in the optical apparatus causing eye strain, digestive derangements.

8. *Electrical* and other conditions of the atmosphere produce headache in certain persons.

Diagnosis of the Cause.—This is often a matter of great difficulty, sometimes all that can be attained is a provisional diagnosis. In the solution of the problem we first consider the age of the patient and the diseases common at the time of life, the circumstances preceding the attack, the time of day at which it occurs, the character of the pain—continuous or paroxysmal—and its duration. The locality of the pain may give some guidance, but it often gives none. We then make a systematic examination of the special senses and of other organs which might produce reflex irritation, also of the blood. The temperature and other symptoms enable us, as a rule, at once to put aside specific fevers and encephalitis, although occasionally in typhoid fever headache and debility are the symptoms which cause the sufferer to seek advice.

Age.—When we have to do with young children we should examine the gums. If the mouth be moist and the child do not suffer increase of pain when the gums are pressed on, dentition is not at fault. We should inquire as to accident, for headache may recur long after injury and in the absence of external mark. Examine the sutures for evidence of hydrocephalus. If the head be drawn back think of basal meningitis. In tubercular meningitis pain is not an early symptom; when it occurs it is in paroxysms, the child screams, may put its hands to the head, there is vomiting, constipation, marked leucocytosis, and the eyes if they can be examined show evidence of optic neuritis. Always when you suspect pain in the head of a child examine the ears for evidences of inflammation of them, by making gentle traction on the auricles, pressing on the tragus; and if possible examine the meatus and membrana tympani. In older children examine the ears, ascertain whether there has been discharge from the meatus; pain from inflammation of the meatus or of the middle ear is relieved after discharge, if not relieved is there abscess? Absence of tenderness on tapping the cranium, and behaviour of the temperature will guide. During second dentition caries and impaction of teeth are possible causes, the latter specially if the jaw be small. Lauder Brunton states that in every case of headache he first examines the teeth. If the child be of school age the condition of the optic apparatus should be examined as to errors of refraction and of powers of fixation, the nose and particularly the naso-pharynx are likely causes at this age. The attention should be specially directed to the latter if there has been frequent ear trouble, examine the naso-pharynx with the finger. If these and other physical causes such as irritable stomach and bowels can be excluded, ascertain the amount of school work which is required. If sleep be disturbed and the child awake unrefreshed, it has probably too much work. Overpressure is a relative term.

The period of eruption of the wisdom teeth is a very variable one, and the irritation connected with the process is a common cause of headache. Megrim often commences about puberty, and the history of the attack is diagnostic, unless in very mild cases. In later life it might be confounded with gouty headache, but the latter is usually confined to one side of the head, is much aggravated by movement; megrim usually becomes less severe in late life, the head may or may not be tender on tapping, there is no history of periodicity, but there is a history of gout in the individual or family, and there may be other evidences of gout present. About middle life a very common cause of headache is commencing presbyopia; if there be increased ocular tension we should be suspicious of glaucoma. In later life a not uncommon cause is uræmia. In every case of headache the urine should be examined.

The Time of Day.—If headache comes on in the morning examine for uric-acidæmia, ascertain whether the bedroom be properly ventilated, and whether the drainage be good. Headache which occurs on awaking in the morning, and is relieved after breakfast, is often due to want of food, nothing having been taken since late afternoon, and then only a light meal. Some congestive headaches are aggravated by the recumbent posture with low head, and are therefore worst in the morning. When the attack comes on in the evening it is often caused by overwork, exhaustion, nerve debility, sometimes by commencing presbyopia. Those which come on or are worst at night are due to rheumatism or syphilis. When the latter is the cause the pain is more constant and the nocturnal exacerbations are, as a rule, more severe than in rheumatism. The headache of rheumatism may be very severe, but it is not so constant as that due to syphilis or tumour; there is usually the history of a draught, sometimes great tenderness of the

scalp, and there is the history of former rheumatic ailments. Occasionally when the structures at the back of the head are affected in rheumatism the slightest movement of the head is painful. The diagnosis of intracranial tumour in early stages presents considerable difficulty, specially if it be slowly growing and if it do not involve any trunk of a peripheral nerve. The difficulty is all the greater from the fact that occasionally hysterical symptoms are the most prominent features in the early stages of organic disease. The characteristics of headache produced by tumour are, the constancy of pain with severe exacerbations, which may come on at night but are not confined to that period, and the pain seems to be present even during sleep. Gowers lays it down as a rule that headache which prevents sleep is due to organic disease, but the rule is not an invariable one. If in such a case examination of the fundus of the eye reveal symptoms of intracranial pressure, tumour may be diagnosed. Hæmorrhage as a cause of headache is excluded by the history. Abscess is often difficult of diagnosis, history of disease of ear, bone, or of pyæmia, with variable pulse and temperature, should guide. In encephalitis severe headache is the first symptom, it increases from hour to hour, and the temperature is elevated. It is not always easy to diagnose hysterical headache; usually the description given is out of proportion to the evident condition, the patient sometimes smilingly descants on the suffering; a common seat of this headache is the vertex; the pain is likened to the driving in of a nail. Violent exercise, laughter, coughing, blowing wind instruments, stooping aggravate congestive headache. If the condition be produced by fatigue, noise, bustle, study, it is probably due to debility or exhaustion. The amount of debility may be gauged by the facility with which the pain is induced,—the more readily induced the more tedious the recovery. If a nursing mother complain of headache after having given the breast to her child she should at once wean it. If headache be produced by sightseeing we have to do with a person debilitated, or naturally delicate, or with eye trouble.

Occasionally the condition of the pulse will put us on the track of the cause. If it be one of high tension, examine for toxæmia, uræmia, uric acid, or some other form autogenetic or heterogenetic. Liver derangement is a not uncommon cause of such trouble.

The *locality* to which pain is referred is not of much aid in diagnosis. Headache from eye strain, although often frontal, may be situated in any part of the head. Pain from ear trouble may be in any part of the same side, even on the opposite side; the ear itself may be free. Removal of cerumen has cured pain over the whole head. Ocular headache is common in biliousness, also in gout. Frontal headache is common in malaria, in dyspepsia, in congestion of the frontal sinuses, in catarrh and influenza, and in inflammations and suppurations of these sinuses. Lauder Brunton states that disease of the nose is liable to cause pain in the frontal region at the margin of the hairy scalp. Pain at, or a sensation of pressure on, the vertex is often complained of in hypochondriasis, hysteria, in women debilitated by prolonged lactation, anæmia, poverty, and about the climacteric. Pain at the back of the head is common in cerebellar tumour, in basal meningitis of children; it occurs also in chronic affections of the nasopharynx. Von Tröltsch, while stating that disease of the naso-pharynx is a common cause of pain in any part of the head, noted that examination of that space with the finger often produced pain at the occiput. Occipital headache occurs in persons who have evidences of nervous breakdown, in constipation, and in rheumatism of the scalp and nucha. Tenderness on pressure and pain on tapping are common in rheumatism of the scalp; the hairy scalp is more liable to be tender than the parts without hair, the vertex more than the occiput. In organic disease pain on tapping or even on pressure over a limited area often indicates the seat of lesion—affection of periosteum or of bone, or a tumour.

Treatment.—The discovery of the cause in many cases indicates the cure. To enter with detail into the treatment of headache would involve the writing of a small treatise on the practice of physic, the number of causal conditions is so great. For the treatment of these the reader is referred to the various subjects—meningitis, anæmia, uræmia, etc. In some cases the first treatment must be palliative, but if in any case the only prescription is antipyrin and rest, it is a practical acknowledgment of failure of diagnosis. In more chronic cases the treatment is at once directed to the cause. For example, if the nose be at fault, the diseased condition must be treated *secundum artem*; if the eye be to blame, the error must be corrected by means of lenses, or prisms, or the necessary combinations of these. Carious teeth may require treatment, and if impacted teeth be the cause the removal of one or more may be necessary; it is often good practice to remove the first permanent molars if they show evidence of caries, in order to obtain room, and often in older persons the wisdom teeth must be extracted. Headache due to periostitis from caries may occasionally be cured by painting the neighbouring gum with equal parts of liquor iodi fortis and of linimentum aconiti; half a minute later rinse the mouth with tepid water, and repeat the painting if required. Occasionally we are called to treat severe headache in association with acute gastric catarrh; nothing gives more rapid relief than a sinapism to the epigastrium and a dose of castor oil. When constipation or

hæmorrhoids are the cause these must have suitable treatment.

In every case of severe headache the patient should be put to rest, with all sources of peripheral irritation removed; ladies often find relief from loosing down the hair. In some fevers and inflammatory conditions the hair should be removed, and cold applied either by means of Leiter's coil or an ice-bag. The ice-bag should not be kept constantly applied, but intermittently; continuous application produces severe pain. Headache due to the congestion of frontal sinuses in catarrh and influenza is best relieved not by cold, but by the frequent application of very hot compresses to the forehead.

In simple *congestive* headache the patient should be put to rest with the head high, a mustard foot-bath, a mustard blister, or turpentine stupe to the nape of neck, a saline aperient, and if the heart be acting violently, small doses of tincture of aconite repeated frequently till relief.

Headache due to chronic congestion has occasionally been cured by free epistaxis, therefore local blood-letting by leeches may in such cases be tried. Occasionally a cantharides blister to the nape of neck has relieved when other measures have failed. Any heart condition causing passive congestion should be treated.

In headache due to *anœmia*, we have also to do with debility of nerve-centres from malnutrition, therefore fatigue and exertion are to be avoided, rest in the recumbent posture favours the free flow of blood to the brain. Arsenic and other treatment for anæmia.

Headache due to exhaustion from fatigue may be relieved by rest, and sipping hot tea, coffee, or soup. Small doses of strychnine or of nux vomica are also useful. If the cause be worry in a nervous individual, 30 grains of bromide of potash, with 4 or 5 minims of tincture of nux vomica, or a dose of bromo-soda (bromide of sodium with caffeine), or a tabloid of phenacetin 4 grains, and caffeine 1 grain, or antipyrin 10 grains. Any of these may be repeated an hour later, if required. Externally, liniment of chloroform alone, or with an equal part of liniment of aconite, may be applied to the painful area.

In nervous debility, in addition to the immediate treatment of headache, diminution of work is necessary; for school children it is often necessary to curtail the amount of work, specially of home preparation. In severe cases complete abstention from it for a long period may be required, change of air and scene, open-air exercise, nourishing diet—fish, egg, and milk. Fellow's syrup or Easton's syrup, with arsenic. Occasionally in such cases, with frequently recurring headache, 10 or 15 grains of bromide of sodium, with tincture of nux vomica, after meals is useful along with some other tonic. Fletcher's syrup of the hydrobromates, with strychnine, is also useful.

In loaded bowels, with liver derangement, a dose of calomel, followed by a purge, gives rapid relief. Some persons who live highly are accustomed to relieve their frequent headaches by blue pill, or calomel, followed in the morning by a saline; the removal of the cause would be better attained by the regulation of the diet and an increased amount of exercise. It is the experience of many medical men, that although mercurials are useful, they favour a recurrence of the morbid condition of liver.

In *urœmic* headache do not give digitalis, but a dose of calomel as a bowel antiseptic, followed by a saline, and at first only water as food. Mustard to the nape of the neck. For the use of blood-letting and subcutaneous transfusion of normal saline solution, see "Uræmia."

In *rheumatic* and *syphilitic* headache, the most useful drug is potassium iodide, 5 to 20 grains three times a day after food. In all cases of chronic headache, even if there be no history of syphilis and no evidence of it, iodide of potash should be tried. In rheumatic headache extract of colchicum, ½ to 1 grain at bedtime, is often effectual, and in rheumatism of the scalp, with tenderness to touch, ammonium chloride, 15 grains in extract of liquorice and water every four hours, often acts like a charm. In gouty headache both potassium iodide and colchicum are useful.

Migraine.—In a considerable proportion of cases of acute headache to which the medical man is called he will find that he has to do with migraine. His treatment must be (*a*) of the attack; (*b*) during the interval. (*a*) Of the attack. The majority of patients have most relief lying down, others must sit; irritation of end-organs is excluded as far as possible, the room is darkened. In mild attacks 30 to 60 grains of guarana powder in infusion gives relief (the tincture is also said to be good); others find that a drachm of bromo-soda cuts short the attack, some use antipyrin, others phenacetin and caffeine. Placing the hands and feet in hot water gives relief in some cases. Some compress the carotids. In some the only means of obtaining relief from the agonising pain is the subcutaneous use of morphine. Such a method should be avoided, if possible. (*b*) Between the attacks. As the nervous system is profoundly involved, endeavour to raise its tone, and avoid everything which might lower it. In some persons uric-acidæmia is a common exciting cause of the attack, therefore in these the diet should be carefully regulated. Haig advises that nitrogenous food should be reduced to a minimum. Some persons enjoy comparative freedom if they abstain from butcher's meat and take only fish. Lauder Brunton advises a nightly dose of sodium salicylate, 15 to 30 grains alone, or with potassium bromide, 10 to 30

grains. Should the salicylate disagree he gives potassium bicarbonate. In some cases the persistent use of ammonium chloride after meals gives freedom. Wilks strongly recommends a nightly dose of cannabis indica, $\frac{1}{4}$ to 1 grain.

Occasionally obstinate headache, which has resisted other remedies, has been cured by the administration of an anthelmintic. In countries in which lumbricoids are common, headache in children is treated with worm nuts; here santonin in castor oil is best. In other cases a dose of ethereal oil of male fern has caused the expulsion of tape-worm, and with it the cure of persistent headache. Sometimes headache has been cured in ladies by cutting the hair short.

In the treatment of chronic headaches many drugs have been used in addition to those already named, both nerve tonics and sedatives. Quinine, zinc, valerian, ergot, butyl chloral hydras, etc. Turpentine was highly praised by Warburton Begbie—20 drops repeated after an hour or two. Balthazar Foster found that nitrite of amyl relieved headache like magic. This remedy and nitro-glycerine might be expected to be useful in the spastic variety (hemicrania sympathetica tonica). Ergot has been recommended in congestive headache, and Campbell recommends that it be tried if other remedies fail. Several writers advise the use of electricity. Campbell states that it has not received the praise due to it because it is seldom used in temporary and occasional headaches; he has occasionally found the pain of intracranial disease temporarily removed by the constant current.

In all cases, specially chronic cases, we must remember that we have to treat a peculiar nervous system, that it should through hygienic and dietetic measures be braced up, that exercise in the open-air is important, that the cause of headache should, if possible, be discovered and removed, and that as rarely as possible should our treatment be only palliative. Alcoholics and morphine to be used as rarely as possible.

Headless Monsters.—Acephalic twin fœtuses. *See* Teratology (*Placental Parasites*).

Head-Locking. *See* Labour in Multiple Pregnancy (*Complicated Cases, Interlocking*).

Head-Nodding. *See* Head-Shaking.

Head-Shaking.

Head-Shaking and Head-Nodding with Nystagmus in Infants (Spasmus Nutans).

Synonyms. — Head-jerking; Head-rotating; Gyrospasm; Nodding spasm.

Definition.—This is a functional co-ordination-neurosis affecting infants under two years—generally when between four and twelve months old.

Clinical Features.—Its two cardinal symptoms—involuntary nodding or shaking of the head and ocular nystagmus—are usually both present, but either may begin some weeks before the other. Sometimes, however, only head-movements are observed throughout; while in other cases there is passing nystagmus only.

The movements of the head may consist in a simple forward nodding, but lateral or rotatory skaking is more common. They cease when the child is lying down, and also when the eyes are closed voluntarily or artificially. The nystagmus is rapid and of short range. It is oftenest horizontal, but may be rotatory or vertical; in the latter case the upper eyelid may participate in the movement. It is generally more marked in one eye than in the other, and it is common to find one eye only affected. Occasionally there is rotatory or vertical nystagmus of one eye, and distinctly horizontal movements of the other. When the head is passively steadied by the hand the nystagmus increases; or it may become visible then for the first time.

Rhythmical contraction and dilatation of the pupil (hippus) may sometimes be found, and occasionally convergent strabismus develops. The child has often a peculiar trick of turning his head to one side and staring fixedly out of the opposite corners of his eyes. This gives him, at times, a curious vacant preoccupied look. The intellect, however, is not at all affected however severe or long-continued the symptoms may be. When the child's attention is attracted in any way this seems to increase the movements. They diminish with drowsiness and cease during sleep.

Course and Duration.—The patients are generally weakly children—very often rickety—and in unsatisfactory hygienic surroundings. The symptoms usually begin suddenly, almost always in mid-winter. They seldom last less than six weeks, usually from three to six months, and sometimes longer. Occasionally, after complete recovery, there is a return during the following winter.

Etiology.—There are many factors contributing to the causation of this condition. Falls on the head and the irritation of teething have been blamed. The most important influences, however, in determining the onset of the disease are the age of the patients, the absence of sufficient sunlight in their surroundings, and the presence of rickets. It is also probable that anything else which temporarily or permanently lowers the vitality predisposes to its occurrence and to its long duration.

Diagnosis.—Cases of spasmus nutans are usually easy to recognise. Eclampsia nutans, or the "salaam convulsion," has sometimes been mistaken for it, but is quite different, being a form of epileptic seizure associated with serious cerebral defect. It is also not difficult to distinguish these cases from those in which

somewhat similar head-movements are due to chronic hydrocephalus or to some other organic brain disease, and in which the prognosis is naturally altogether less favourable. The nystagmus of these cases can often be distinguished from more serious forms by its having recently set in without obvious cause, and by the movements being confined to one eye or being different in the two eyes.

Treatment.—While sedatives may appreciably diminish the extent of the movements they are not of much importance in the treatment. The chief thing is to secure abundance of fresh air and sunshine, and to put the child on an anti-rachitic regimen with cod-liver oil and phosphorus.

Healing Sore. *See* Ulcers and Ulceration (*Healing Sore, Treatment*).

Health. *See* Epidemiology; Meteorology (*Influence on Health*); Vital Statistics (*Test of Health of a Community*).

Health Resorts. *See* Therapeutics (*Health Resorts*).

Hearing, Artificial Aids to. *See also* Auditory Nerve and Labyrinth; Brain, Physiology of (*Motor Areas, Eighth Nerve*); Brain, Tumours of (*Localising Symptoms*); Brain, Cysts (*Porencephaly*); Children, Development of (*Hearing*); Deaf-Mutism; Ear, Examination of, etc.; General Paralysis (*Symptoms*); Mind, Education of (*Memory*); Paranoia; Stomach and Duodenum, Diseases (*Remote Symptoms*); Uræmia (*Acute, Special Senses*).

The artificial aids to hearing may be divided into two groups, those which by their action improve the impaired conductive function of the diseased ear, and those which, by collecting the waves of sound, bring them to bear upon the organ in a more concentrated form. To the former group belong the varieties of so-called "artificial tympana." The latter group comprises the various forms of ear-trumpets, conversation-tubes, etc. A knowledge of their action and limitations is necessary to every practitioner, since the exploitation of useless and often harmful artificial aids is one of the commonest of the many forms of quackery which are the curse of this country.

1. *Artificial Tympana.*—A nearly complete list of the different artificial ear-drums invented by surgeons is as follows:—1. Yearsley's cotton-pellet. 2. Turnbull's cotton-ball attached to thread. 3. Turnbull's disc of adhesive plaster. 4. Turnbull's disc of sublimated gauze. 5. Thomas' disc of oiled silk. 6. Barr's cotton-pellet fixed by collodion. 7. Blake's disc of sized paper. 8. Field's disc of india-rubber, cotton-wool, and flannel. 9. Burkhard Merian's solid disc of india-rubber. 10. Toynbee's solid piece of india-rubber on a silver wire stem. 11. Politzer's india-rubber tube as long as the meatus. 12. Lucæ's india-rubber disc attached to a rubber tube. 13. Downie's circular patch of egg pellicle. 14. F. H. Pierce's disc of stout linen with cotton thread soaked in vaseline. 15. Richardson's gold cylinder. 16. Michael's glycerine thickened with tannin. 17. Farquhar Matheson's plug of powdered boric acid. 18. Ward Cousin's hat-shaped membrane of compressed cotton. 19. Dadysett's combined trumpet and ear-drum.

Of these artificial tympana, Toynbee's instrument and all its modifications are to be condemned. They are the favourite pattern of the aural quack, and, at the meeting of the British Medical Association in 1888, the opinion was expressed that the so-called "ear-drums" largely advertised, and consisting of a piece of india-rubber fixed to a metallic stem (Toynbee's form), often cause serious injury to persons already suffering from defective hearing, especially in those who are the subjects of suppuration.

The variety most employed by otologists is the original cotton-pellet of James Yearsley, first introduced into practice in 1848. This simple contrivance owed its origin to the following occurrence:—In 1841 a gentleman from New York consulted Yearsley for large perforations of both membranes. On being told the condition of his ears, he said: "How is it, then, that by the most simple means I can produce in the left ear a degree of hearing quite sufficient for all ordinary purposes?" He did this by means of the "insertion of a spill of paper, previously moistened at its extremity with saliva, which he introduced to the bottom of the passage."

Yearsley's artificial tympanum consists of a small pellet of cotton-wool, either twisted up with a tail, or tied about its middle with a cotton thread. Hartmann, of Berlin, prefers a pellet with an elongated tail, the latter tied round with thread, and the whole waxed.

The pellet probably acts as its inventor originally believed, viz. by supporting the ossicular chain. Yearsley believed that the membrane has an important action in supporting the ossicular chain, and that the pellet restored this support when lost by perforation. In some cases of old non-suppurative middle ear inflammation with a lax membrane the cotton-pellet will greatly improve the hearing power.

The advantages which may be claimed for Yearsley's cotton-pellet are:—(1) It is simple and effective. (2) The patient can make it for himself. (3) It can be made of antiseptic wool. (4) It is easy of manipulation. (5) It is well tolerated by patients (one of Knapp's cases wore one for 29 years). In fact, if the writings of modern otologists be consulted, it will be found that it is this, or some modification of

this form of "artificial drum" only that is of any practical use.

The cases best suited are those in which the membrane is partially or totally lost, but it will be found that in all varieties of perforation, from small holes to almost complete loss of the membrane, there will be cases in which the application of the artificial drum is attended with marked benefit, while in others apparently similar no good can be obtained even after long perseverance. It is especially where there is a solution of continuity in the ossicular joints (particularly in that between the incus and the stapes) that the pellet will be of service.

When the malleus and incus are present the pellet requires to be placed in contact with them. In cases where the head of the stapes is free the wool must be placed against that ossicle. In the latter case, however, a tenotomy of the stapedius will often give better results, since, when the stapes is free from its connection with the incus, the stapedius tilts it out of place and the pellet acts by preventing this.

The presence of profuse discharge is a contra-indication to its use.

The application of the cotton-pellet is quite simple. Made of suitable size of sterilised or antiseptic wool, it should be moistened with glycerine of carbolic acid and passed steadily down the meatus until the membrane is reached. This is done by means of a pair of fine straight forceps (Yearsley designed a special pattern for the purpose), and seldom causes pain if gentleness is used. The hearing should be tested previously, and the pellet gently adjusted until repeated tests show improvement. The adjustment is often tedious, but is speedily learned afterwards by the patient, who should be taught to manipulate the aid himself. He should be directed to wear the pellet for an hour or two only at first, gradually increasing the time until it is worn continuously. It should never be worn at night.

2. *Ear-Trumpets, etc.*—A large variety of trumpets, conversation-tubes, and other instruments have been devised and introduced from time to time as aids to hearing. Amongst them the aural quack finds a happy hunting-ground, and advertises freely his cornets, auricles, aerophones, apparitores auris, and similar valueless arrangements to catch the money of the unwary. These quack instruments may be at once dismissed from consideration, they are almost always useless, many cause considerable irritation and even ulceration of the meatus, and the smaller ones may sometimes be found in contact with the membrana tympani bathed in the foul pus of a suppuration caused by their presence. The "akouphone" and "massicon," recently forced upon the public in a very plausible manner, are equally useless and fraudulent.

The special function of the auricle and meatus is the collection and reflection of the sound waves, whereby as few of them as possible are lost. The natural and fundamental form of artificial "ear-trumpet" is the placing of the hollowed hand to the ear. This augments resonance and increases the auditory capacity, especially for notes of high pitch and short wave-length, such as are found in the overtones of the human voice. All the trumpet forms of artificial aid are designed to act similarly. They are made of various substances—ebonite, iron, glass, porcelain, silver-plated metals, etc., the best reflectors being those of hard and dense material. Against their employment, however, their weight, fragility, and intrinsic notes greatly militate, and therefore such light metals as aluminium are often used. The simplest (and, therefore, the best) form is that of a hollow cone, and this, modified in various ways, is that usually adopted for trumpets. Echoes may be lessened by widening the cone-base and obliquely truncating it. Some years ago Dr. C. J. B. Williams published the result of certain experiments with these aids to hearing. He found that the confusion of transverse vibrations could be obviated by perforating the sides of the trumpet. He pointed out that a cone of stiff paper, some eighteen inches long, ending in a short metallic ear-piece, caused very little reverberatory roar, and magnified sound twelve times. The writer on one occasion found that such a contrivance, home-made, was the only form which benefited one particular patient.

The modifications of these aids are so numerous that space will not allow of any complete description of them, and, indeed, such description would not serve any useful purpose. Otologists are practically unanimous in the opinion that, with the exception of conversation-tubes and simple forms of trumpet, these aids to hearing classed in this second group are useful in but a small proportion of cases. Reference to instrument makers' catalogues will give some idea of the multiplicity of form amongst these aids.

A few words must be said regarding "audiphones" before discussing tubes and trumpets in more detail. The audiphone is a fan-shaped disc of vulcanized rubber, bent by a silken cord. The edge is placed in contact with the upper teeth, the convex surface being turned toward the source of sound. The waves of sound caught thereon are conveyed to the internal ear by bone-conduction, *via* the upper teeth. The contrivance will, however, be found of little value, save in a few exceptional cases. With them, as with all "aids," it must be remembered that the presence of an intact (or nearly intact) perception of sound is necessary if one hopes to obtain any improvement by augmenting sound, collection, and conduction.

From what has been already said, it will be

gathered that of this class of artificial aids to hearing only two forms are of actual practical value :—

(*a*) The conversation-tube.
(*b*) A simple form of trumpet.

(*a*) *The conversation-tube* consists of a trumpet-shaped mouthpiece, to collect the sound waves, connected with a metal or vulcanite earpiece by means of a piece of tubing of covered rubber strengthened by spiral wire. The mouthpiece is held by the person with whom communication is required, who speaks into it in ordinary tones. Here it may be remarked, that in conversing with deaf persons a distinct enunciation in ordinary tones will always be heard the best. The greater part of the sound in spoken words is produced by the vowels, and it is the consonants which form the distinguishing part of words and which have the softest tone. The louder one speaks, the more intense is the sound of the vowels as compared with the consonants; hence in shouted or loudly-spoken words the former are apt to eclipse the latter, and the distinctiveness of the words is thereby impaired or lost. Shouted conversation is therefore worse heard than that uttered in ordinary tones.

The conversation-tube can be worn round the neck or carried coiled up in the pocket, and is decidedly superior for near conversation.

(*b*) A simple form of *trumpet* is required for hearing at a distance ; that which is, *par excellence*, the best is the one known as the *London horn*. It is made in three sizes, and should be painted a dull black, the plated ones being somewhat conspicuous. It consists of a bell-shaped receiver, from the side of the large end of which springs a curved tube, ending in an ivory, ebony, or vulcanite earpiece. The mouth is usually covered with a fenestrated disc. The earpiece is introduced into the meatus, the large open end being directed towards the source of sound. This instrument is excellent for churches, concerts, lectures, theatres, and the like. A noticeable drawback to the metal horns lies in the metallic adventitious sounds which they convey, particularly when listening to singing, orchestral music, etc.

Among other artificial aids to hearing may be mentioned the "rod-ostephone" of Thomas, and the modification thereof of Cresswell Baber. These are used after the manner of the "audiphone" already described, and aim at transmitting sound through the teeth and the bones of the skull. Their use, however, is very limited.

At one time much was expected of the microphone as an aid for the deaf, but hitherto all results from that invention or its modifications have proved impracticable and disappointing.

To sum up the subject, therefore, it may be said that of all the artificial aids to hearing that have hitherto been devised, only three are of any real practical value, namely, a simple and unirritating form of "artificial ear-drum," the conversation-tube, and the London horn.

It is well to advise patients who wish to use a trumpet or tube not to purchase in too great a hurry, but to obtain several forms on approval or hire in order that they may, by experiment, discover the one which suits them best before purchasing.

Heart. *See* Adrenal Glands, Adrenalin (*Uses, Heart Disease*) ; Alcohol (*Morbid Changes in Chronic Alcoholism, Heart*) ; Alcohol (*Indications*) ; Alcoholism (*Chronic, Complications*) ; Anæmia (*Symptoms*) ; Anæsthesia, General Physiology ; Anæsthesia, Chloroform (*Objections, Heart Failure*) ; Aneurysm (*Signs and Symptoms, Heart-Sounds*) ; Angina Pectoris ; Balneology (*General Balneo-Therapeutics, Affections of Cardio-Vascular System*) ; Beriberi (*Symptoms, Cardiac*) ; Chest, Clinical Investigation of the (*Heart*) ; Chest, Injuries of (*Wounds of Heart*) ; Children, Clinical Examination of (*Circulatory System, Heart*) ; Digitalis ; Hæmatemesis (*Causes and Source*) ; Heart, Embryology, Comparative Anatomy, and Physiology ; Heart, Affections of Myocardium and Endocardium ; Heart, Neuroses of ; Heart, Congenital Malformations of ; Heart, Surgery of ; Hydatid Disease (*Special Organs, Heart*) ; Labour, Diagnosis and Mechanism (*Fœtal Heart*) ; Labour, Management of (*Anæsthetics, in Heart Disease*) ; Lung, Tuberculosis of (*Etiology, Predisposing Factors, Heart Disease*) ; Lung, Tuberculosis of (*Complications, Endocarditis*) ; Medicine, Forensic (*Injuries, Ruptures, Heart*) ; Meninges of the Cerebrum (*Purulent Meningitis, Causes*) ; Pericardium, Diseases ; Pharmacology ; Physiology, Circulation (*Heart*) ; Pleura, Diseases of (*Acute Pleurisy, Signs of Effusion, Displacement of Heart*) ; Pleura, Diseases of (*Pneumothorax, Clinical History*) ; Post-mortem Methods (*Examination of Cavities, Thorax, Heart*) ; Pregnancy, Physiology (*General Changes, Circulation*) ; Pregnancy, Diagnosis (*Objective Symptoms and Signs, Fœtal Heart Sounds*) ; Pregnancy, Intra-uterine Diseases (*Fœtal Endocarditis*) ; Puerperium, Physiology of (*Changes in Maternal System, Circulatory System*) ; Puerperium, Pathology (*Ulcerative Endocarditis*) ; Puerperium, Pathology (*Sudden Death, Causes*) ; Pulse ; Syphilis (*Tertiary, Pathology, Vascular System*) ; Therapeutics, Health Resorts (*Climate and its Effects*) ; Visceral Pain (*Hollow Muscular Organs, Heart*) ; etc.

Heart — Embryology, Comparative Anatomy, and Physiology of.—It is necessary to the understanding of diseases, disorders, and malformations of the heart, that we should bear in mind the

origin and development of the organ, as well as particulars in its comparative and essential anatomy and physiology. It is, therefore, proposed to state as clearly and as shortly as possible such points in the embryology, comparative anatomy, essential structure, and functions of the normal organ, as are calculated to explain its behaviour when diseased, disordered, or malformed.

The Embryology of the Mammalian Heart.—Of the three layers of the germ, the ectoderm, the mesoderm, and the endoderm, whence all parts of the organism are derived, it is the mesoderm which contributes chiefly to the formation of the heart. The endocardial lining of the organ is alone derived in all probability from the endoderm, which, however, as the "endothelial heart," is a very distinct structure in early fœtal life. The cerebro-spinal nerves, which ultimately penetrate and permeate the organ, are alone derived from the ectoderm, while recent research has confirmed the correctness of Remak's belief that the sympathetic ganglia spring from the mesoderm like the muscular, vascular, parenchymatous, and pericardial constituents of the heart (Paterson, *Phil. Trans. B*, London, 1890).

While these three sources are welded into the unity of a whole in this organ, as throughout the body, there is thus a largely preponderant community of origin between the muscle, vessels, sympathetic nerves, parenchyma, and pericardium of the heart.

The embryonic heart in the mammal is at first a bilateral tubal organ placed at the cephalic extremity of the medullary groove, which coalesces by fusion into a single tube, and finally emerges from development as the unified though bipartite heart of completed mammalian growth. At a very early stage the embryonic heart, which continues throughout essentially a single tube, acquires the property

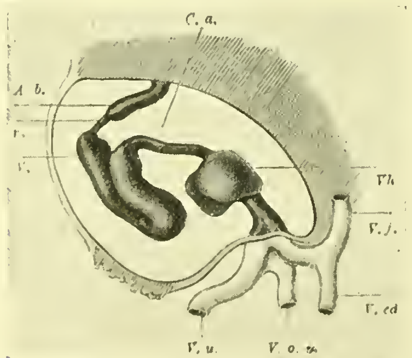


Fig. 1.—The endothelial heart seen in profile (His). *A.b.* aortic bulb; *r*, fretum Halleri; *v.* ventricle; *C.a.* auricular canal; *V.j.* jugular vein; *V.cd.* cardinal vein; *V.o.m.* omphalo-meseraic vein; *V.u.* umbilical vein.

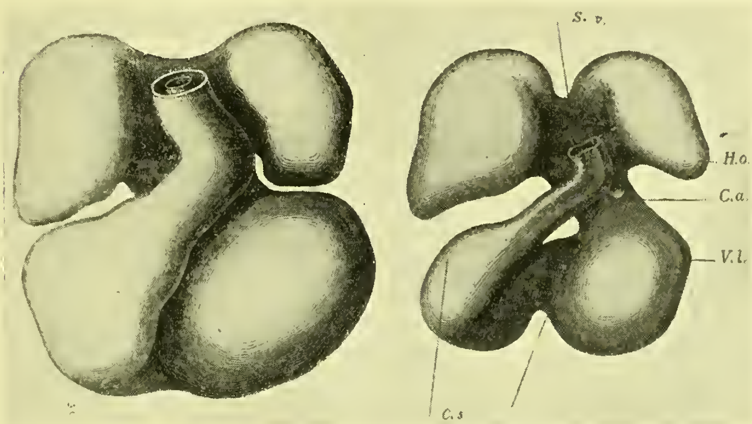
of rhythmical pulsation. As it grows lengthwise it accommodates itself to the somewhat confined space destined for it, by bending upon itself. Early in development, also, the heart of the amniote embryo, whether human or other, not only exhibits this accommodative bending, but also shows variations in its diameter which indicate its division into auricle, ventricle, aortic bulb, and the channels which unite these portions. The earliest indication of the cardiac apex is given by the bending and close apposition of the ventricular portion of the tube, best seen in the inner or endothelial heart, while as yet the auricular and aortic portions are at some distance from the ventricular, and separated from it by the auricular canal and so-called fretum Halleri. These connecting channels are the sites of the future valves. With this continued growth of the organ the auricular portion assumes a bipartite configuration, and of this portion of the still single chamber the right division is the larger. A similar indication of division in the ventricular portion is now seen, and of these still communicating divisions the left is the larger. At this stage the aortic bulb turns towards the middle line and upwards, so that the rest of the aorta proper comes to lie between the two auricles.

With these variations in external configuration certain internal changes are gradually associated. Septa arise from the roof and floor of the auricular portion, which ultimately completely divide this part into two distinct chambers. A septum likewise springs from the floor of the ventricular portion, which finally erects a complete partition and brings into being two distinct ventricles. With this septal division, a valvular demarcation has gradually associated itself, and the chambers of the once common cavity are shut off, not merely auricle from auricle and ventricle from ventricle, but the corresponding chambers to the right and left from each other by means of the auriculo-ventricular valves.

While the heart is still a comparatively undifferentiated tube, its aortic bulbar end is in connection with a bilateral system of five aortic arches, the obliteration of some portions of which and the persistence of others finally constitute the adult mammalian arterial system. The obliteration of the right and left first and second arches, and of a portion of the stem between the third and fourth, leaves the permanent channels of the internal and external carotid arteries; while the obliteration of the whole of the right fifth and of the stem connecting it with the right descending aorta, leaves the fourth right arch to constitute the right subclavian artery and its branches. The fourth left arch remains as the permanent transverse and descending aorta, while the fifth left arch forms the ultimately obliterated *ductus arteriosus*. The persistent stems between the right and left third and fourth arches remain as the common carotid arteries. From a septation and division

of the aortic bulb the pulmonary arteries come into being.

At the caudal end of the heart certain changes occur in the embryonic venous system,



FIGS. 2 and 3.—Muscular and endothelial heart of the embryo (His). *S.v.* sinus vestibuli; *H.o.* auricle; *C.a.* auricular canal; *V.l.* left ventricle; *C.s.* conus arteriosus.

which result in the persistent veins. In the pre-placental embryo venous blood is collected from the vascular area, and brought into the body by two large vitelline or omphalo-meseraic veins, whose destiny is to become the portal venous system of the adult. With the appearance of the placenta blood is brought from that important organ by two umbilical veins ultimately fused into one in the cord, but persistent for a time thereafter as two in the abdominal cavity. The umbilical and vitelline veins, together with the duct of Cuvier—a trunk which collects the blood from the upper or jugular section of the primitive venous system and the lower or cardinal—open into the right and left venous sinuses which are continuous with the auricular heart.

These sinuses ultimately coalesce, and the dextral entrances constitute the inlets of the permanent systemic venous system, while the pulmonary veins, after the formation of the lungs, enter the sinistral portion of the sinus.

We have seen that in the formation of the ultimate arterial system a larger portion of the right than of the left aortic arches is obliterated in the course of development. In the early bilateral venous system the reverse is the case. In the establishment of the persistent veins a larger portion of the left than of the right primitive system is obliterated, except in rare and exceptional instances. The arteries, like the projected arterial stream, grow into the body; the veins, like the returning venous current, grow in a measure out of it. Thus, with the formation of an obliquely transverse communication between the upper part of the left primitive jugular vein and the lower part of the right corresponding vessel, the current from the lower portion of the vessel on the left is in great measure diverted from it, and the channel shrinks finally to obliteration, with the exception of a portion at its lower end which persists as the coronary sinus, and a part at the upper end which remains as the superior intercostal vein. The transverse branch, and the cause of this diversion of current, becomes the left innominate vein, the future important recipient of the chief lymph stream from the thoracic duct. The relation of the thoracic duct and vein is of practical moment in retrograde stasis due to heart disease.

A similar but reversed process meanwhile takes place between the left and right cardinal veins. An obliquely transverse communication is established between the lower portion of the left and the upper part of the right cardinal vein, and persists as the vena azygos minor. It coalesces with the right cardinal vein to constitute the larger azygos vein. The confluence of the left and right innominate veins thus constitutes the mighty stream of the superior vena cava, which is further swollen before it

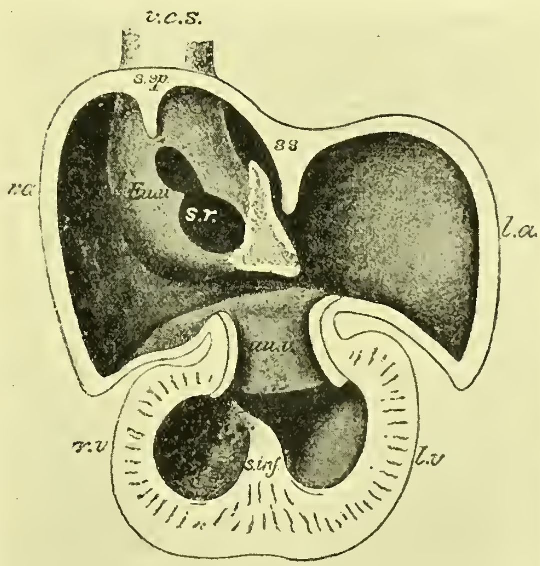


FIG. 4.—Septatim of the heart of the human embryo (Quain after His). *V.c.s.* vena cava superior; *s.sp.* septum spurium; *s.s.* septum superior; *Eu.v.* Eustachian valve; *s.r.* sinus venosus; *r.a.* right auricle; *l.a.* left auricle; *au.v.* auricular canal; *s.inf.* septum inferior; *r.v.* right ventricle; *l.v.* left ventricle.

debouches into the right auricle by receiving the vena azygos major. The veins from both sides of the body have meanwhile coalesced more or less in the middle line to form the inferior vena cava.

The important remaining and intervening portion of the venous system is completed, as has been stated, by the changes which take place in the development of the vitelline or omphalo-mesenteric and the umbilical veins, which result in the formation of the portal

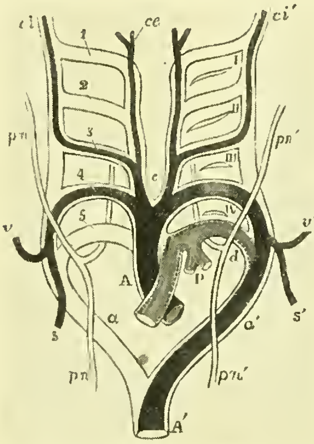


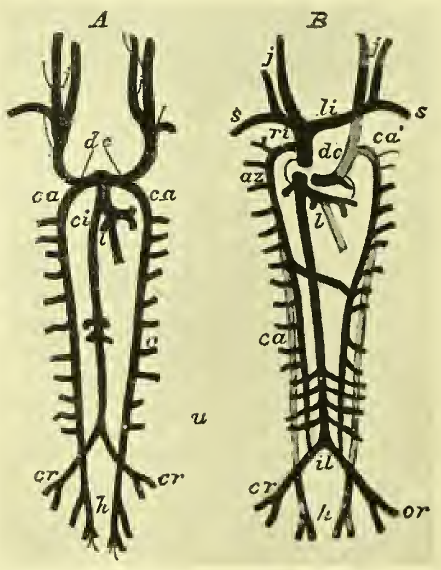
Fig. 5.—Diagram of the transformation of the aortic arches into permanent arteries (Quain after Rathke). A, P, ascending aorta and pulmonary arteries; 1, 2, 3, 4, 5, right branchial anterial arches; I, II, III, IV, left branchial clefts with arches corresponding to those on the right; *ci*, *ci'*, right and left internal carotid arteries; *ce*, external carotid arteries; *c*, common carotid arteries; *v*, *v'*, vertebral arteries; *s*, *s'*, subclavian arteries; *d*, ductus arteriosus; *a*, *a'*, right and left aortic roots; A', permanent descending aorta; *pn*, *pn'*, right and left pneumogastric nerves. Obliterated portions left unshaded.

venous system and its supra-hepatic outflow, the great hepatic vein.

The great hepatic and umbilical veins are further united during later fœtal life through the medium of the ductus venosus, which, like its arterial analogue, the ductus arteriosus, disappears after birth. The disappearance of these channels coincidently with birth is, however, due to opposite causes. The ductus arteriosus disappears, because the breathing organ of adult life—the lung—has come into action; the ductus venosus vanishes because the breathing organ of fœtal life—the placenta—has ceased to exist.

Notwithstanding the confluence of the two great venous channels, the upper and lower venæ cavæ at the right auricle, there is, during the greater part of fœtal life, little admixture of the two streams. The difference in direction of these, the large development of the Eustachian valve at the entrance to the inferior cava, together with the direction given by the auricular septum and its deficiency at the foramen ovale, serve to direct the inferior blood-stream—that most oxygenated by placental respiration—across the right into the left auricle, and thence by way of the left ventricle and the arteries of the aortic arch into the upper part of the body; while the blood returning by the superior cava to the heart, and therefore the less oxygenated, passes through the right auricle into the right ventricle, and is sent thence chiefly by way of the ductus arteriosus into the abdominal aorta and the territory irrigated by the latter. The situation of the ductus arteriosus or Botalli and its entrance into the aorta beyond the primary vessels of the aortic arch is a beautiful provision of Nature for supplying the most nutrient fluid to the organs most necessary to fœtal life, namely, the heart, by way of the coronary arteries and the growing nerve-centres indispensable to organic life.

While a portion of the pulmonary circulation is probably pervious *in utero*, and circulates the less oxygenated blood contained in the right ventricle, the effect of the respiratory act at birth is to open out the whole of the pulmonary blood-vessels, and thus to accommodate a large quantity of blood. Pressure in the left auricle is thus suddenly raised, the foramen ovale is closed by its valve-like screen, a change in the position of the heart is brought about, which, according to Schantz (Pflüger's *Archiv f. d. gesammte Phys.* Nov. 1888), twists to some extent the ductus arteriosus, and the circulation then assumes the character of that of adult life.



Figs. 6 and 7.—Diagram of the condition of the great veins in the embryo, and of their transformation into the permanent veins (Quain after Kölliker). *j*, Jugular veins uniting with the subclavius: *s*, *ca*, cardinal veins becoming the azygos as in B; *dc*, ducts of Cuvier formed by the union in BA of jugular and cardinal and becoming the superior vena cava on the right side, but disappearing on the left side; *ca'*, part of left cardinal vein which disappears; *ci*, vena cava inferior; *l*, hepatic veins and ductus venosus; *il*, common iliac veins; *cr*, external iliacs; *h*, hypogastric becoming the internal iliacs. Portions which disappear less darkly shaded than the permanent portions.

The fœtal channels are, as a rule, altogether obliterated within a period variously estimated from a week to ten days.

The Comparative Anatomy of the Mammalian Heart.—Professor Huxley has shown (*Man's Place in Nature*, Edin. and Lond. 1864)

how the human fœtus in its development is an epitome of the process in vertebrates lower than man in the scale of being. He also suggests that the unity of the process may extend to still lower creatures. Support is found to the truth of this contention by a comparison of the development and anatomy of the heart of the higher vertebrates already given, with the characters and conditions of the heart in the non-vertebrate and lower vertebrate animals.

In the lowest organisms which take in solid food the nutritive material is mainly distributed from the digestive spaces directly into the protoplasm of the body (*Elements of Comparative Anatomy*, by Carl Gegenbaur, translated by F. Jeffrey Bell, p. 50, 1878). The next step in differentiation is the existence of a distinct digestive tube, through the walls of which nutriment passes into the body protoplasm. A little higher and we find the mid-substance or mesoderm of the organism containing a cavity—the cœlum—distinct both from its digestive tube and outer layer or ectoderm. The cœlum contains a fluid which changes its position during alterations in the configuration of the organism as a whole. Gegenbaur suggests that this is to be regarded as the first indication of a circulating nutritive fluid (*loc. cit.*). Later, canalicular cavities regularly arranged form the prototype of a vascular system, which a little later still open into a perienteric space, and we then reach the confines of the completely shut off heart and vascular system of vertebrates. This progress in development is well shown by a series of vascular apparatus figured by Gegenbaur to illustrate the circulation in Mollusca (*op. cit.* p. 368). These show the transition from a rhythmically contracting tube sending its contents anteriorly and posteriorly, to one receiving fluid posteriorly and transmitting it anteriorly as in the mammalian heart, the receiving cavities being the prototypes of the auricles, the transmitting of the ventricles. Between these two divisions and distinguished by the direction given to the circulating fluid, there is the remarkable phenomenon evinced by the Tunicata of a rhythmical vascular system propelling its contents for a time entirely in one, and then after a short pause by a process of reversed rhythmicality in the opposite direction (Gegenbaur, *op. cit.* p. 406).

It is, however, the persistent characteristics of the heart in the craniote vertebrates which have most interests for students of the circulation and its disorders in man, inasmuch as some of these offer us types of those malformations which occur in the fœtal heart from arrest in development of, or occlusions taking place in portions of the heart, during the various phases of its growth.

The determining factor in the development of the heart in the Craniota appears to be the situation of the organs in which blood already used by the body is aerated for further use. When such organs, as in the case of fishes the gills, are, so to speak, outside the body, that is, directly exposed to external aerating conditions, it is manifest that the oxygenated blood returned to the body requires only a single-chamber system for its reception and propulsion within the body. We find, therefore, in fishes, that the heart consists of a single auricle and ventricle. In the amphibia, the characters of the heart in fishes persist to the same extent as the subaqueous habits of the amphibian. The nearer members of this class are to the fish in habits the more do their hearts resemble that of the fish. That is, although the auricle is divided more or less by bands, the two auricles, if they can be so regarded, still communicate freely, and in addition to the ventricle is even less divided. It is, indeed, practically one chamber. The efferent tube is still an aortic or arterial bulb, a branchial system of arteries still exists, as well as pulmonary arteries, and, finally, a ductus arteriosus establishes communication between the pulmonary artery and aorta, as in the case of the mammalian fœtus.

The heart of the reptile shows a farther step in differentiation. The auricles are distinct, as also in great measure are the ventricles. In the crocodile the ventricular partition is complete. In those members of the group in which this separation is not perfect, as in the snake and the turtle, the communicating deficiency at the base of the ventricular septum is so obstructed by a strong muscular valve-like structure as to leave arterial blood in the left and venous blood in the right chambers of the heart. The blood is, moreover, directed by this arrangement, the arterial into the systemic arteries, and the venous into the pulmonary vessels. In some persistent malformations of the mammalian heart the reptilian heart is well represented. In birds, as in mammals, which exist chiefly in air (for the diving bird and the diving mammal (whale) must rise to breathe) the auricles and ventricles are completely divided, and the chambers are more capacious than in creatures lower in the scale of being. The difference between birds and mammals is rather in the character of one of the valves than in the chambers of the heart. The right auriculo-ventricular orifice in the bird is guarded by a strong muscular falciform fold or curtain. This is attached to the anterior wall of the right ventricle, and follows pretty accurately the curve of the conus arteriosus. The right ventricle as a whole is small, and is wrapped round the large and powerful left ventricle. The physiological significance of these conditions is interesting, and probably related to the regulation of the venous supply to the lungs under the very varying conditions of pulmonary inflation in flight and in repose.

Many of these conditions in the lower animals are, as we have seen, represented, during one period or another of development, in the higher mammals, including man. The external respiratory organ of the mammalian fœtus, its gills so to speak, is the placenta, and the corresponding cardiac condition is but a more complex form of the simple single-chamber system of the fish, which gradually increases in complexity to that of the more differentiated reptile, and ultimately reaches the perfect division seen in the heart of the adult air-breathing animal. In no process, indeed, is seen more perfectly than in the development of the mammalian heart the evidence of what the older writers called "design in nature," and it is difficult to use any other term even now. The adaptation up to a certain point of existent organs to present circumstances is a well-recognised fact in vital processes, whether physiological or pathological; but the preparation and perpetuation of organs and organisms for future use, and to meet conditions foreseen and intended, but not yet present, appear to some to be beyond the power of a mere acquired habit of growth or haphazard fusion of species. The controversy is, after all, however, but a war of words, for the results of "natural selection" are quite the same as those of appropriate combination, and the determination of causes from effects opens up a chain of reasoning one end of which, at least, is lost in infinity, and cannot be wholly explained by physiology alone. (For particulars concerning the anatomical situation of the heart, the reader is referred to the section on Clinical Investigation of the Chest, vol. ii. p. 83.)

The Physiology of the Mammalian Heart.—If the heart of a mammal be dissected after having been boiled sufficiently, it will be found that the arrangement of its muscular fibres has an important relation to its action as a vital organ. In the first place, the auricular portion of the heart may be detached from the ventricular, and the dividing line between these, which occupies the place of the auricular canal and fretum Halleri in the fœtus, is seen to consist of fibrous material of a more or less resistant character, which surrounds in great measure the auriculo-ventricular and arterial orifices of the heart, and enters into the constitution of its valves, tendons, and interstitial tissue. In some large animals, indeed, a portion of this dividing line situated between the aortic and the two auriculo-ventricular orifices becomes fibro-cartilaginous and even bony. In the next place, the arrangement of the muscular fibres of both auricles and ventricles shows that the organ in its growth has been at first bilateral and then unified. That is, there are fibres which seem to appertain to each half of the auricular and ventricular portions of the heart, and others which embrace both the right and left auricles or ventricles as the case may be. In the third place, the muscular fibres of the external and middle portions of the ventricular wall appear to be continuous with those which pass into the ventricular septum and papillary muscles (Pettigrew, *Phil. Trans.* 1864; Quain's *Anatomy*, vol. i.).

There is also some muscularity of the cardiac ends of the two venæ cavæ and of the pulmonary veins, which in quadrupeds at any rate is most marked in the case of the superior cava. Muscular elements are likewise said to enter into the constitution of the auriculo-ventricular valves (Foster).

Cardiac muscular fibre is of the striped variety, but the striæ are not so distinct as in skeletal muscle, and suggest a transition condition between the latter and unstriped visceral fibres.

The important physiological consequence of these arrangements is, that separate and consecutive action of the auricles and ventricles is secure, and that all parts of these chambers act simultaneously during the period of their respective contraction, and relax at the same time during their quiescence. As a succinct description of the cardiac cycle nothing can be added to the following lucid statement:—

"When the chest of a mammal is opened and artificial respiration kept up, the heart may be watched beating. The great veins, inferior and superior venæ cavæ and pulmonary veins, are seen, while full of blood, to contract in the neighbourhood of the heart: the contraction runs in a peristaltic wave towards the auricles, increasing in intensity as it goes. Arrived at the auricles, which are then full of blood, the wave suddenly spreads at a rate too rapid to be fairly judged by the eye, over the whole of these organs, which contract with a sudden sharp systole. In the systole the walls of the auricles press towards the auriculo-ventricular orifices, and the auricular appendages are drawn inwards, becoming smaller and paler. During the auricular systole the ventricles may be seen to become turgid. Then follows, as it were immediately, the ventricular systole, during which the ventricles become more conical. Held between the fingers they are felt to become tense and hard. As the systole progresses the aorta and pulmonary arteries expand and elongate, the apex is tilted slightly upwards, and the heart twists somewhat on its long axis, moving from the left and behind towards the front and right, so that more of the left ventricle becomes displayed. As the systole gives way to the succeeding diastole the ventricles resume their previous form and position, the aorta and pulmonary artery shrink and shorten, the heart turns back towards the left, and thus the cycle is completed" (Foster, *Text-Book of Physiology*, Part i. p. 232, London, 1893).

The length of time during which these events occur is estimated by Foster to be about 0·8

second, of which he apportions 0·3 second to ventricular systole, and 0·5 second to complete ventricular diastole which includes 0·1 second for auricular systole. In these calculations the heart is assumed to maintain an average rate of 72 beats to the minute (*op. cit.* p. 264)

With these movements of the heart two sounds and two pauses are associated. The first, a long, booming sound, occupies a considerable portion of ventricular systole, and is most intensely heard near its commencement; the second, a short, sharp sound, occurs during a small portion of ventricular diastole at its commencement. Between the first and second sounds there is a short pause difficult of measurement, and between the second and following first sound a considerable and more easily appreciable pause not audibly broken by the systole of the auricles. These facts may be represented in the diagrammatic manner first employed by Sir William Gairdner of Glasgow, and which has been variously modified by subsequent writers.

The explanation of pauses in action is of course easy. Absence of action is of necessity as noiseless as death itself. The short pause ensues when ventricular systole is spent. It is virtually the commencement of ventricular diastole, which is only broken by a brief passive event,—the closure of the semilunar valves, which emit a short, sharp sound on closing under the influence of blood-pressure in the aorta and pulmonary artery. The cause of the first sound has been much debated, and an examination of the method of its production has a practical interest. It may be heard when the thorax is opened artificially, and also when the heart is exposed by congenital defect in the chest wall. It cannot, therefore, be due to the heart being contained in the thorax, or to the impact of its apex against the chest wall. The events accompanying it are: (1) systole of the ventricles; (2) opening of the semilunar and closing of the mitral and tricuspid valves; (3) the pressure of blood upon all the constituents of the chamber which contains it; (4) the rise of this pressure over that of the column of blood contained in the efferent arteries; and (5) the consequent passage of blood into these. Foster (*op. cit.* p. 240), without entering into details, states that if the ventricular portion of the heart be cut off below the level of the auriculo-ventricular valve, the detached portion may be heard to emit sound. If this be so, it is highly suggestive of muscular contraction playing a part in the production of the sound, but that part may be a very small one. As regards the obscuration of a sound or sounds of the heart by defect in its valvular apparatus, careful clinical observation in many cases and in all varieties of valvular disease has convinced the writer that such obscuration is in direct proportion to the amount of noise generated and not to the extent of the lesion present in any particular valve. He has also known the heart-sounds to be clearly audible without murmur when both aortic and mitral valves were much diseased and the heart beating very feebly at a rate of 150 in the minute. The clear sounds probably arose in this case in the right ventricle, which was ascertained to be sound at the necropsy. In determining experimentally the rôle of the valves in the production of the first sound, not only one but all four would have to be placed out of action during cardiac systole. This has never been done so far as the writer is aware.

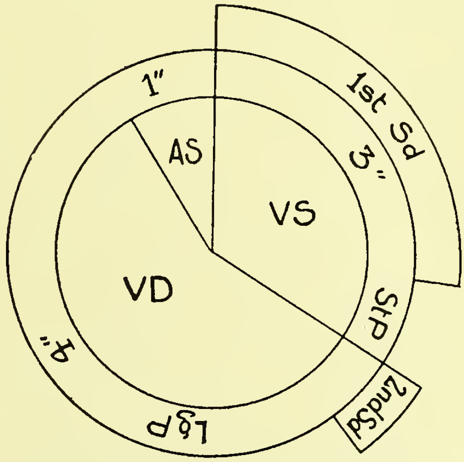


Fig. 8.—A.S., auricular systole; V.S., ventricular systole; V.D., ventricular diastole; 1″, 3″, 4″, notation of time occupied by phases in cardiac cycle; St.P., short pause; Lg.P., long pause; 1st Sd., first sound; 2nd Sd., second sound.

Although Dr. Halford (*The Action and Sounds of the Heart*, p. 25, 1860) believed that the first sound of the heart was due to tension and vibration of the auriculo-ventricular valves during ventricular systole, he proved on several occasions, by careful experiment before capable and credible witnesses, that when the systemic venous inflow was prevented by forceps, and in addition the flow from the pulmonary veins by grasping them between the fingers, sound ceased to be emitted during systole, and returned immediately blood was again allowed to enter the heart. Drs. Yeo and Barret (*Journal of Physiology*, vol. vi. p. 145) repeated Halford's experiment, but without grasping the pulmonary veins at the root of the lungs. They found that under these circumstances cardiac systole was still accompanied by some, but greatly diminished sound. Dr. Arthur Leared argued and endeavoured to prove that the cause of sound during systole was not in the apparatus of circulation, but in the blood itself, and believed that the impact of the moving ventricular blood against the more stationary aortic blood was the essential cause of the first sound of the heart (*Essay on the Sounds caused by the Circulation of the Blood*, London, 1861).

Finally, Sir Richard Quain (*Proc. of the Royal Soc.* June 1897) expressed the opinion that the first sound of the heart was the result of the ventricular blood being projected by a spiral movement, as shown by Professor Pettigrew, against the aortic cusps held down by the aortic column of blood. This view he had held and expressed in 1852, but did not publish till recently. The difference between it and Dr. Leared's is only that Sir Richard Quain attached some importance to the intervening aortic valves as a factor. In performing the experiments on which in part his conclusions were based, he used an apparatus constructed out of a sheep's heart, in which the right chambers were cut away, the coronary arteries ligatured, and the mitral valves destroyed. A column of water was then made to press upon the aortic valves and the ventricle was also filled with water. When now systole and diastole were imitated by alternate compression and relaxation of the ventricle by the hand, a very good imitation of both sounds could be elicited. He was good enough to ask the writer's assistance in the performance of this simple experiment, and the latter can add his testimony to Sir Richard Quain's as regards the facts.

Taking all these data into consideration, it appears legitimate to conclude, that more than one factor enters into the production of the first sound, and that the chief place is to be assigned to the resistance offered by aortic blood-pressure to the ventricular blood-pressure when the latter is suddenly and forcibly rising above it. If this conclusion be accepted, it follows that the audibility and character of the first sound may, *ceteris paribus*, afford some evidence of the vigour of ventricular contraction.

The Coronary Circulation.—The nourishment of the mammalian heart by blood is chiefly effected by the coronary arteries, which spring from the aorta behind its two anterior semilunar cusps. The pulse in all arteries is systolic in time, and the coronary arteries are no exception to the rule. There is reason, however, to believe that the repletion of the aorta on cardiac diastole also imparts additional impulse to the coronary arterial circulation, while the strong valve in the coronary sinus prevents regurgitation from the right auricle. Sibson (*Medical Anatomy*, p. 73) observed that both arteries and veins on the surface of the heart became turgid and tortuous during systole, and again straight during diastole. This fact appears to indicate that when ventricular contraction is at its height, some pressure is exercised upon the smaller arteries and upon the venous outflow from the coronary sinus. The anatomical situation of the coronary vessels in the texture of the heart is admirably calculated, however, to reduce injurious compression or obstruction to a minimum, as may be seen from the following illustration.

Anastomosis between the two coronary arteries has been disputed by some anatomists, but appears to have been proved to exist by Dr. Samuel West (*Lancet*, June 1883, p. 945) by injected specimens. It is probable, however, that it is not very free, a circumstance for which the original bilaterality of the organ in embryo may in a measure account. The most important and most constantly active organ in the body thus receives a constant supply of recently oxygenated blood, a circumstance which, when associated with an otherwise normal constitution of the blood, appears to be a leading factor in the maintenance of a healthy state and normal action of the cardiac muscle.

We have now briefly examined the muscularity and vascularity of the heart, and the chief physiological questions of practical interest associated with these factors. It remains to examine shortly the third factor in the production of the functional unity of the heart's action.

The Cardiac Nervous System.—The nerves of the mammalian heart spring from the trunk of the vagus and from the inferior cervical ganglion of the sympathetic chain. The trunk of the vagus, however, is a mixed one, and contains fibres of the spinal accessory nerve which avoid the jugular ganglion on the vagus, and passing through its lower ganglion or "ganglion of the trunk," course to the heart in the vagus. These are said to be of small calibre like all efferent visceral spinal nerves, according to Gaskell (*Journal of Physiology*, vol. vii.). The sympathetic spinal nerves of the heart are derived from the upper dorsal spinal nerves from the second to the fourth and perhaps the fifth, but chiefly from the second and third (*see* Plate, p. 154). The gathering point for these in the thoracic chain is the stellate or first dorsal ganglion, issuing whence they encircle the subclavian artery as the so-called ring of Vieussens, and thence by way of the lower cervical ganglion pass to the heart. Both vagal and sympathetic branches from either side having reached the organ contribute to the superficial and deep cardiac plexuses. Here in all probability an incomplete peripheral decussation occurs, so that the nerves from either side supply both halves of the heart or both aspects of the unified tube of which the heart consists. This seems to be proved by the fact that unilateral section of the nerves does not materially disturb the heart's action, while bilateral section soon proves fatal. The majority of the medullated fibres of the vagus lose their medulla in the cardiac plexuses, the majority of the medullated fibres of the spinal cardiac nerves in the ganglia of the sympathetic chain which intervene between their point of issue from the cord and their exit from the inferior cervical ganglion. For our anatomical knowledge of these important facts we are largely indebted to Gaskell of Cambridge and his successors in this country and abroad. Kölliker of Würzburg affirms his

belief (*Gewebelehre*, p. 858) that nerves which retain their medulla in the visceral periphery are afferent and therefore sensory, as on their way from the cord or medulla they pass through or over intervening ganglion without being broken up by them. On the visceral distribution of efferent branches of both the vagal and sympathetic series ganglion cells occur, and as the function of these two streams differs, their ganglion cells must remain essentially as distinct as the nerves themselves, however agglomerated. The final distribution of the nerves to the visceral muscle is by fine nucleated plexuses, which end upon muscle cells both cardiac and vascular. In the opinion of the majority of present-day histologists they do this by so-called free ends, the existence of which a minority still disputes or considers doubtful. Such an arrangement, however, would seem to be necessary to the exercise of the separate functions of the different nerves. The muscle cell appears to be the term common to both, and the probable medium of interchange between them, by processes too little known for fruitful discussion at present.

By means of this mechanism the regulated action and nervous nutrition or trophation (to coin a convenient but not very euphonious term) of the heart is secured. Although the embryonic heart has a rhythmical action before it is supplied either with organised vessels or with nerves, the conditions of sustained rhythmicality seem to require an integrity of all three factors —the muscle cell, the blood which bathes it, and the nerves which regulate it, and in some obscure way exercise a trophic influence upon it. By means of the nervous system the heart may be retarded or even arrested in action, that is, inhibited; quickened in rate and in force or augmented; and weakened, or depressed. The channel for inhibition, depression, and trophation is the vagus; for augmentation the sympathetic spinal nerves. The spinal accessory fibres already mentioned are now regarded as the inhibitory fibres, and are efferent in action *towards* the organ. The depressor fibres exercise their influence *from* the heart by way of both ganglia on the vagus at the vaso-motor centres in the medulla, whence they induce a fall in the peripheral blood-pressure. They are therefore afferent or sensory in character.

The activity of trophic nerves, of which we are justified in assuming the existence, is probably efferent. Sensory and motor fibres likewise exist in the sympathetic spinal nerves and constitute the peripheral mechanism of augmentation. They are, indeed, believed by some to be the chief seat of cardiac sensibility, and the character and distribution of radiated pain in sensory disorders of the heart support this view. The general effect of retardation of the heart's action is regarded as conservative of energy or anabolic; that of acceleration or augmentation as expending energy and leading to exhaustion or catabolism. It has been maintained by some that the existence of efferent ganglia on the trunks of visceral nerves, and the interposition of the sympathetic system between the cerebro-spinal centres and the viscera, may be the cause of the differences between somatic and splanchnic nervous action, whether affecting the heart or other involuntary organs. The whole subject is, however, far from clear at present, but a good deal has been done to elucidate it during the last ten years or so. Like the unification of the heart itself, the unity of the cerebro-spinal and sympathetic systems has been secured by a process of fusion, for there is good evidence to show that in their essence they are embryologically distinct, and spring from different layers of the germ, as has been stated at the commencement of this article. Although, finally, the sympathetic system is now considered to be wholly or almost wholly dominated in function by its cerebro-spinal connection, the question of a retention by it of a measure of efferent autonomy cannot yet be regarded as settled.

Heart, Affections of Myocardium and Endocardium.

General Introduction—

The first part of this article consists of a consideration of all the various etiological factors, pathological processes, and their cardiac and systemic effects, followed by a description of the recognised types of heart disease.

Secondly: The symptoms produced by heart-failure irrespective of its special cause are considered, and certain peculiarities associated with special forms of disease are referred to.

Thirdly: The physical signs of the different varieties of heart disease are described in detail, and their common combinations according to the pathological state that gives them origin are sketched.

Fourthly: The treatment—hygienic, dietetic, and medicinal—of heart disease is considered.

It is evident that a pathological knowledge should precede the study of symptomatology, physical examination, and the principles of treatment, inasmuch as these last three can only be profitably studied in the light of pathological experience. As regards their *symptomatology* we are entitled to consider nearly all the forms of heart disease together, seeing that they all tend to, and generally eventuate in, that condition we term *venous stasis*, in which the blood accumulates, as it were, on the venous side of the circulation, giving rise to the ordinary symptoms to be described, while stasis of blood in the chambers of the heart is the common cause of the embolic process and its clinical manifestations. If, as physicians, we are ever to keep before us the "tendency to death" in the case we are treating, then in heart disease the expression of that "tendency" is *venous stasis*.

It is by *physical examination* and the signs thereby elicited that *differentiation* of the various forms of heart disease is accomplished.

But as regards treatment, inasmuch as it is to the heart muscle practically alone that our efforts are directed, it is allowable again to generalise, and to consider together all varieties of heart disease.

The pathological considerations that precede symptomatology, etc., will amply demonstrate the importance of obtaining in every heart case an accurate account of the patient's previous health, for without this it is impossible to interpret aright either the symptoms or the signs, to forecast the probable course that will be run, and to treat the patient with the best prospect of success.

A. GENERAL PATHOLOGY

I. Etiology

1. Exciting Causes.—These are best considered according to the *nature* of the excitant.

(1) *Mechanical.*—The cardio-vascular apparatus has long been viewed as a machine, and cardiac physics have very rightly become a recognised branch of physiological study. But while it is necessary to avoid the fettering influences of a mere mechanical conception, the action of physical agencies must be admitted as producing or inducing temporary derangements and sometimes establishing permanent defects.

Occasionally excessive exercise or unwonted exertion in a subject absolutely or virtually sound seems to act on the cardiac muscle or the aortic segments as an acute strain. Even trained athletes after exceptional physical effort have manifested evidences of cardiac derangement. Not a few cases are on record where in seemingly healthy adults after some unusual effort physical examination has clearly demonstrated a condition of cardiac dilatation. Prolonged exercise, such as is illustrated by some athletic sports, is peculiarly liable to exert strain on the right ventricle, but it must be admitted that such most usually occurs in the untrained or those constitutionally untrainable and unfit for athletic pursuits. The effects of excessive physical effort or ill-regulated drill on young recruits, and the consequences of forced marches even on seasoned men, is well known, and "soldier's heart" has become a well-defined clinical entity. As might be expected, the effects of mechanical influences are most frequently met with in men, and particularly during the years of greatest activity and physical vigour.

The consequences of violent effort are to be clearly distinguished from those of chronic strain. Many laborious occupations necessitate the maintenance of a state of persistent or long-

continued exertion. Porters, navvies, miners, blacksmiths, workers in iron and other heavy metals, and like laborious occupations, are all liable to cardiac derangement and failure from the unavoidable strain associated with their work.

The influence of physical effort in the production of an attack of angina pectoris, when the heart is ill-nourished by an imperfect blood-supply, is so well recognised as hardly to need mention. The relation of muscular effort to blood-pressure may undoubtedly become of pathological importance, especially in subjects who have passed the meridian of life, which period, however, is not to be stated in years. In advanced life nature demands and usually ensures that there shall be a tempering of the strain to the weakening powers.

It must, however, be admitted that in the majority of cases the effects of "strain" do not generally make themselves apparent unless there is also some other agent or agencies at work impairing the cardiac nutrition, or in some way rendering the tissues more vulnerable. In many instances careful investigation will show the patients to have been the subjects of anæmia, chronic alcoholism, toxæmic conditions, or nutritional derangements. In some instances an inherited cardio-vascular weakness may play an important part in precipitating the breakdown. The effects of fatigue during periods of convalescence, overwork at times of physical depression, and ill-regulated exercise in the presence of actual cardiac lesion, are only too frequently met with to permit of denial.

Strain is often said to be accountable for lesions of the aorta. Undoubtedly prolonged effort, such as is necessarily associated with certain laborious occupations, plays a very important part in the establishment of forms of chronic aortitis, particularly the variety usually described as "atheromatous," which, moreover, often implies interference with the coronary orifices and impaired nutrition of the heart muscle.

To sudden strain is also due the rare laceration of a degenerate myocardium, and possibly an acute dilatation of one or more of the heart's chambers.

It is sometimes said that external violence is capable of producing rupture of a cardiac valve. Without absolutely denying this, the extreme rarity of such an event warrants one in believing that it only occurs when the valve is already the seat of some morbid process, and even then it is extremely uncommon to meet with any such condition as could be considered due to external violence. Injury sufficiently severe to rupture a sound valve is almost invariably immediately fatal.

Latent disease is frequently made manifest by some form of undue or unaccustomed exercise. Not infrequently chronic cardiac pathological conditions are much aggravated by unwonted resort to procedures necessitating ill or unregulated effort. A hitherto efficient compensation may be completely ruptured by a trifling accident or a slight tussle. The importance of such considerations on therapeutical measures and medico-legal investigations will at once be evident.

It is not necessary to discuss here the particular localisation of the effects of strain. It will be sufficient to point out that those pernicious mechanical influences which, collectively, we may include under "strain" exert their power (i.) by establishing functional derangement (including leakage of valves and deficient emptying of chambers), which may be merely temporary or prolonged until organic defect is established; (ii.) by producing arterial valvular defects either as the result of dilatation of the aorta and its orifice, or through changes in the valves themselves.

(2) *Chemical.*—Agents conveyed in a soluble form by the blood to the various structures of the heart are accountable for many of the morbid conditions which form the pathological basis of cases of so-called cardiac failure, which, coming under the observation of the physician in their advanced and final stages, present features more or less in common, and principally indicative of waning muscular powers. Certain of these chemical bodies are of autogenetic origin, as in Bright's and Graves' diseases, while others are heterogenetic, being manufactured outside the body, and wilfully or accidentally introduced. The action of toxic agents resulting from microbial growth will best be referred to later.

Among the foremost of the toxic bodies produced within the system must be mentioned those associated with the condition of *gout.* The influence of gout in producing functional derangement of the heart has long been recognised, but its most prejudicial effects are expended either directly or indirectly in establishing changes, usually leading to increased cardio-vascular pressure, and generally associated sooner or later with cardiac hypertrophy. Gout also seems to lead to degenerative changes in the coronary vessels, thereby producing an impaired nutrition of the muscular substance of the heart. The cardio-vascular associations of "gouty" kidney are not to be overlooked. It is very probable also that gout is capable of establishing primary degeneration in the muscle itself. Some of the cases of gouty syncope are probably due to such myocardial changes. It is sometimes said that gout is accountable for a form of chronic mural endocarditis, but this, if it ever occurs, is quite exceptional. The establishment of an aortic valve disease (apart from mere aortic dilatation) is more probable.

Of even greater importance than gout stands out *rheumatism.* It is unnecessary here to consider whether the toxins associated with this

disease are of microbial origin or dependent on morbid activity of the cells of the body; suffice it to say that rheumatism has a particular affinity for the serous membranes of the heart, and the majority of cases of diseased pericardium and endocardium are due to its influence. But rheumatism also very clearly exerts its influence on the muscular substance of the heart, and this seems to be particularly the case in young subjects. In not a few cases of acute rheumatism without endocarditis, or any exceptional degree of temperature, cardiac dilatation ensues apparently from the direct paralysing and degenerative influence of the rheumatic poison on the protoplasm of the muscle cells.

In many of the so-called constitutional diseases cardiac affections arise, but in many instances it is impossible to distinguish between those dependent on impaired nutrition and those arising from toxic influence.

First amongst the pathogenic chemical agents of extrinsic origin must be placed *alcohol*. This body, especially in the form of beer, is accountable for a large number of cases of muscle failure occurring in adult labourers. The most characteristic cases of "alcoholic heart" are certainly met with in excessive consumers of malt liquors, or those who take their drinks "mixed," and where enormous quantities of drink are imbibed in the day while engaging in work of a heavy, often irregular and straining character. Some have thought that local differences in the manufacture of malt liquors may account for the seeming greater frequency with which alcoholic hearts are met with in some districts. Certainly in Manchester "alcoholic hearts" formed a very large proportion of the cases of primary muscle failure, and occurred particularly amongst market porters, barmen, brewers' draymen, cabmen, and labourers. The arsenical contamination of beer which prevailed extensively in Manchester and neighbourhood was, no doubt in part, answerable for many of these cases of muscle failure. "Alcoholic heart" is not commonly met with in women, who are less exposed to strain. It is interesting to note that athletes have long realised that beer was bad for their "wind."

Reference may here be made to the cardiac depressant action of certain animal and vegetable poisons and medicinal substances, such as thyroid preparations and numerous other well-known drugs. Suprarenal extract, however, leads to the development of high arterial tension. The deranging influence of tobacco is too widely experienced to need more than mentioning.

(3) *Thermal*.—Little need be said respecting the influence of heat and cold, light and darkness, on cardiac action and cardio-vascular structure. Exposure to extremes either of heat or cold produces a paralysing influence on muscle. Heat syncope and cardiac collapse from sunstroke are well-recognised clinical conditions. Pyrexia exerts a distinctly deleterious influence on cardiac muscle. The effects of cold in raising vascular tension must also be borne in mind.

(4) *Biological*.—Parasitic agents are very influential in establishing certain well-defined cardio-vascular lesions. The most striking example is "malignant,' or, as it is now frequently termed, "infectious" endocarditis. Some pathologists look upon all rheumatic lesions of the endocardium as due to microbial infection. Inflammatory affections of the heart, particularly those of the pericardium and endocardium, occasionally occur in the course of scarlet fever, diphtheria, and sometimes in some of the other infectious diseases. Most if not all of the septic diseases seem capable of producing secondary affections of the heart.

The embryos of certain of the worms occasionally attack the heart. The cystic form of Tænia echinococcus may produce "hydatids" of considerable dimensions.

2. Predisposing Influences.—These may be considered according to whether they act through (*a*) the inherited or acquired textural characters of the individual, or (*b*) by modification of the environment.

(1) *Heredity*.—Certain families manifest a distinct tendency to early degenerative processes in their vessels, and to a premature cardiac deterioration. It is not necessary to discuss to what extent this is due to direct transmission. In some cases community of work and environment is a more reasonable and likely explanation. Family proneness to gout and rheumatism, and other conditions peculiarly liable to exert a baneful influence on the heart, must not be lost sight of. Cardiac failure from "vascular" causes oftentimes seems to "run" in families. As far as can be ascertained, race exerts comparatively little direct determining influence.

(2) *Sex*.—This is a factor of considerable importance, although the precise way in which the influence may be exerted is in many instances far from clear. The influence of occupation is closely allied with that of sex. Aortic disease is thus most frequently met with in men. Inefficiency of the aortic valves when occurring in females is almost always due to organic changes following rheumatic endocarditis or consequent on microbial infection. Mitral stenosis is much more frequently met with in women than men. Some have thought that this arises from a greater liability to rheumatism and chorea in the female. Primary muscle failure is generally met with in men. "Alcoholic heart" is rare in women. Dilatation from muscle failure and high arterial tension is, however, not unusual in women after the climacteric. Dilatation of the heart from chronic bronchitis and emphysema would seem to be equally common in the two sexes. The importance of chlorosis in young

girls, in producing minor degrees of cardiac dilatation, must not be overlooked. In exophthalmic goitre or Graves' disease, most common in females, tachycardia and irregular cardiac action may be followed by evidences of dilatation and muscle failure. In modifying or increasing already existing physical cardiac deficiencies, the influence of pregnancy on the circulation, including increased arterial tension, must be remembered. Malignant endocarditis has been known to follow parturition.

(3) *Age or period of life* is a factor of great importance. Three periods may be recognised :—Firstly, that of *development* (including fœtal life), where maldevelopment and inflammatory processes along with their consequences may be expected. Secondly, the period of *maturity*, when the effects of strain and the influence of toxic agents, especially alcohol, are liable to be manifest, and when also the mechanical deficiencies of previous valvular involvement are apt to become apparent. Thirdly, the period of *decline*, when affections arising from malnutrition associated with vascular disease and degenerative changes are found.

Of diseases peculiarly prone to affect young subjects, and at the same time leave permanent crippling of the heart, rheumatism stands pre-eminent. Rheumatic fever and rheumatism in "silent" and easily overlooked forms, constituting little more than "growing pains," are very common in early life. This is also the period for the incidence of chorea, which is very often associated with endocarditis, and not infrequently with some degree of myocardial involvement.

Scarlet fever, and possibly some of the other infectious fevers, may exceptionally inflict injury on the endocardium in childhood. It would also seem as though a rheumatic endocarditis might be the first and only manifestation of the rheumatic poison. Speaking broadly, pericarditis and endocarditis are peculiarly the cardiac affections of the developmental period of life. The older a person grows the less liable is rheumatism, even though it occur, to inflict serious injury.

In adolescence, when activity and instability are striking features in the organism's development, so-called functional derangements readily arise.

In the period of adult life, as already indicated, the effects of strain on a healthy or already crippled heart, either from improper, unsuitable, or excessive work or ill-regulated exercise, commonly become manifest. In certain classes of the male community "alcoholic heart" is apt to be developed. The effects of other toxæmic states may become manifest, particularly those resulting from gout and Bright's disease. Syphilitic disease of the aorta and forms of atheromatous aortitis may by encroachment on the orifices of the coronary arteries or extension to the aortic cusps give rise to anginal attacks or the signs of aortic incompetence.

In the female, at both ends of the period of sexual activity, functional derangements are common. In chlorotic females a certain degree of cardiac dilatation is frequently detected. The influence of pregnancy, parturition, and lactation must not be lost sight of, especially in the case of a heart in any way crippled.

Mitral stenosis is the common cardiac lesion of adult females. Conditions of mitral regurgitation through a dilated auriculo-ventricular orifice, as the result of a rheumatic valvulitis, are not met with in either sex so commonly as was formerly believed, and certainly cases of mitral regurgitation through a dilated orifice with sclerosed segments are but rarely seen in the post-mortem room. The possible existence of mitral stenosis should always be carefully considered in every female presenting evidences of mitral incompetence and having an old rheumatic history.

In the advanced stages of middle life a tendency to general adiposity becomes apparent, and in many instances enormous deposition of fat in and around the heart impedes its action and impairs its nutrition, and not infrequently is associated with distinct degenerative changes which may lead to actual dilatation of the ventricular cavities.

In the later years of life processes of degeneration and infiltration lower the vitality and physical powers of both heart and vessels. Arterio-sclerosis paves the downward path. The coronary arteries are frequently the first to suffer, thereby diminishing or cutting off an adequate nutrition of the essential structure of the heart. Hence follow atrophy, fatty degeneration, fibrous transformations, and possibly aneurysm, softening, and it may be rupture of the ventricular walls.

In old people, although by no means limited to them, chronic Bright's disease often leads to associated cardiac hypertrophy, which sooner or later gives place to dilatation and its consequences.

In the years of diminishing vigour atheroma of the aorta is common, and either by extension of the atheromatous process to the aortic valves, or from enlargement of the aortic ring as a result, dilatation of the aorta and incompetence of the aortic valves may be produced. In cases of this kind the atheromatous condition may have extended to the coronary vessels, or at least have narrowed their orifices, thereby impairing the nutrition of the myocardium, and rendering adequate compensatory hypertrophy of the left ventricle imperfect or of short duration. At this time of life anginal attacks may occur.

(4) *Temperament.*—It is very difficult to decide to what extent "the sum of the physical

peculiarities of an individual, exclusive of all definite tendencies to disease," should be allowed a place in the etiology of cardiac derangements. Temperament is not an easily estimated factor. Nevertheless in functional derangements of the heart it cannot be altogether laid aside, and even in organic disease has to be remembered when arranging a course of management.

(5) *Previous Disease.*—A former morbid process may (i.) by affection of the structure of the heart itself or (ii.) by producing impairment of tissues outside the heart have rendered the cardio-vascular apparatus more vulnerable or less able to meet the needs of the body and the requirements of daily life.

Some previous pathogenic influence may have led to injury, or established a process which, even when the particular etiological agent is withdrawn, leaves a textural scar or functional weakness which makes the affected tissues peculiarly liable to capitulate.

The most influential agent is rheumatism. This, especially when acting in the form of "rheumatic fever," commonly produces endocarditis, and frequently pericarditis. One attack is commonly followed by others, and whilst the heart is perhaps most liable to be damaged in the first, each subsequent exacerbation or fresh establishment of endocarditis renders the affected parts more predisposed not only to similar attacks, but to succumb to other pernicious influences. Thus in the case of "malignant" endocarditis it will often be found that the valves have been already crippled by a simple rheumatic process.

Scarlatina is said to precede pericarditis and endocarditis; but if all cases of rheumatism in scarlet fever be excluded, one is bound to admit that, considering the frequency of this infectious disease, it can only be accorded a very subordinate place amongst the affections predisposing to distinct cardiac disease.

Measles is also sometimes said to predispose to endocarditis, but this is doubtful.

Reference may here, too, be made to the influence of renal disease, especially when in the form of "granular kidney," in producing extensive cardio-vascular changes. In not a few cases the ventricular hypertrophy ultimately gives rise to a fatal dilatation.

(6) *Climate.* — Indirectly climatic conditions may be of considerable influence in the production and progress of cardiac affections.

The cardio-depressor action of cold is of much importance, and some are still found sufficiently loyal to ancient beliefs as to consider soil, temperature, and humidity important influences in promoting the incidence of rheumatic lesions of the heart. As might be expected, climate is chiefly of importance in cardiac etiology in so far as it is related to rheumatism. Rheumatic fever, however, would seem to be almost ubiquitous. The influence of malaria in the production of cardiac disease must also be noted.

(7) *Social position,* and all therein included, is to be credited with wide reacting influence for weal or woe in the production and development of cardiac disease. It will be sufficient to remind the reader that dilatation of the heart from laborious occupation or excessive beer-drinking, or the two combined, is most frequently met with in the working class. Aortic regurgitation is common in men of all sections of society. It is generally thought that incompetence resulting from a dilatation of the aorta is commonest in working men, while that dependent on valvular sclerosis from the extension of an atheromatous aortitis is common in all ranks of life.

(8) *Education* must be allowed a place in considering the predisposing causes of cardiac disease. Not a few cardiac breakdowns date from a period of ill-advised and badly regulated physical education; especially is this of importance when athletic pursuits are strenuously continued beyond youth and early manhood. The importance of mental strain and emotional stress in the precipitation of functional derangements must not be overlooked.

(9) *Occupation.* — As above indicated, the "calling" in life is often of the greatest influence in determining the special form of cardiac disease.

Occupations of a laborious character will lead to hypertrophy, and if continued in states of malnutrition, with implication of the cardiac vessels, or after the limit of healthy compensation has been attained, dilatation and degenerative changes will inevitably result. The importance of work and pursuits necessitating sudden strain or intermittent stress cannot be over-estimated.

Work requiring exposure to damp and cold and such influences as are supposed to lead to rheumatism indirectly become of some importance in the etiology of cardiac disease.

Duties or avocations making exceptional calls upon the nervous system are liable to produce functional derangements of the heart.

Sedentary workers, who feed not wisely but too well, frequently develop conditions associated with high arterial pressure, sclerotic changes in the coronary and other vessels, and degenerative changes in the myocardium. Ultimately such persons may suffer from cardiac dilatation, and present signs and symptoms practically identical with those so commonly seen in the over-worked and under-fed aged labourer.

(10) *Habits.*—From what has already been stated the bearing of habits on cardiac health cannot be forgotten. Bad habits, hygienically speaking, if they cannot be credited with directly engendering cardiac disease, at least oftentimes go far in exposing the subjects of them to the attacks of definite irritants.

Errors of eating and drinking must be held

responsible not only for such conspicuous conditions as "alcoholic" and "gouty" heart, but for many manifestations of malnutrition and a considerable number of the so-called functional derangements of the heart.

Possibly the use of belts or constricting bodies may have exerted some influence, as was once thought, in the production of "soldier's heart."

Tobacco and indulgence in snuff and certain drugs also exert an indirect influence in deranging the cardio-vascular mechanism.

Reference may here also be made to habits which give rise to states of morbid excitement. In attacks of temper or conditions of worry or fright death may result from angina pectoris and syncope. Protracted emotion also exerts a prejudicial effect upon the heart and vessels. Sexual excesses may prove a not readily recognised cause of cardiac derangement.

II. Morbid Processes

Before describing the individual lesions met with in the more common cardiac affections, it will be desirable to consider briefly the essential nature of the morbid processes affecting the cardiac structures. This constitutes what may be termed the general pathology of diseases of the heart.

(1) *Maldevelopment.*—The normal process of cardiac evolution may be arrested or deranged and malformation or malposition result. These can only be interpreted by a reference to embryology. Although it is manifest that most congenital cardiac deficiencies are dependent on some interference with the development of the fœtus, but little is known respecting the remote etiological factors. The oft-quoted influence of maternal impressions is little more than mythical.

Congenital defects arising from fœtal inflammation are more easily understood. An inherited tendency to rheumatism or rheumatic fever in the mother may possibly lead to the establishment of an endocarditis in the embryo, but here also evidence is very uncertain.

A consideration of the individual forms of cardiac malformation does not come within the scope of the present article, having been discussed elsewhere. It is, however, necessary to observe that at no period of existence can the cardiac textures be considered free from the attacks of pathogenic excitants, or incapable of reacting thereto. In studying the forms of cardiac disease originating after birth, it is necessary to remember that the predisposing influence of a developmental aberration may play an important part. Thus malignant endocarditis not infrequently attacks a heart the subject of some slight anomaly in the number or size of the valves, or in the character of the septum. A hypoplasia of the heart has been claimed as a common association of chlorosis, and explanatory of certain features of the anæmic heart, but it is well to remember that in anæmia the heart is often dilated.

It only remains to add that congenital processes of disease, whether essentially of developmental origin or of the nature of a fœtal inflammation, are capable of initiating a series of conditions which usually declare themselves by such striking features as cyanosis, blood alterations, various nutritional derangements, abnormal cardiac action, and occasionally dropsy.

(2) *Anæmia.*—The integrity of the cardiac muscle is dependent upon an adequate supply of normally constituted blood. Conditions of cardiac bloodlessness may arise from local causes, or be a part of a general poverty of blood. In both cases the myocardium suffers. As it is the office of the blood to convey nutriment to the cardiac textures, states of anæmia if prolonged are apt to lead to degradation of structure and impairment of function.

In simple anæmia the heart may be markedly involved. As already indicated, the cardio-vascular apparatus in chlorotic girls may be in a condition of subnormal development. The cardiac tissues are pale, often soft and flabby, diminished in size and consistency, and microscopically are usually found to be in a state of lowered activity, as indicated by the reaction of the protoplasm of the cells to staining reagents, or by changes indicative of its actual conversion into lower forms. In not a few cases of chlorosis distinct cardiac dilatation occurs.

In pernicious anæmia the regressive changes become conspicuous, and fatty degeneration of the cardiac muscle may be manifest to the naked eye, as the so-called "tabby-cat striation" or "thrush's breast" appearance. This irregular distribution contrasts strikingly with that of a localised anæmia due to limited vascular disease.

Anæmic conditions also tend to produce a condition of hydræmic dropsy, and occasionally not only may slight effusion be met with in the pericardial sac, but a somewhat sodden and œdematous condition of the cardiac textures generally may occur.

Thus from general anæmia and its immediate consequences serious cardiac derangements ensue. Functional deficiencies are common, asthenic manifestations are easily brought out by slight effort, and dilatation of the ventricular cavities readily develops.

Anæmia of local origin is almost always dependent on impairment of the blood-supply from disease of the coronary arteries. At all events from a practical standpoint, the coronary arteries are to be considered as terminal vessels. This seems to be particularly the case as regards the important papillary muscles. Obstruction to the lumen, thickening of the walls, or compression of the vessels from without, inevitably lead to profound nutritional changes which, while starting as a simple local anæmia, quickly

pass to more important states of degeneration or actual necrosis.

Anæmia of the myocardium is also said to occur from the pressure of pericardial effusions, and in conditions of acute and even chronic dilatation, but perhaps generally a state of local cyanosis is more apt to accrue.

(3) *Hyperæmia.* — Little can be said with certainty respecting the process of hyperæmia as it occurs in the cardiac structures, since post-mortem it usually leaves no distinctive characters, and clinically presents no special features. Some clinicians, however, believe that some of the temporary or so-called functional derangements of the heart may possibly be due to a state of local congestion. At the present time considerable doubt exists as to the occurrence of any condition meriting the term of general hyperæmia or plethora, although formerly many cardio-vascular derangements were so explained.

Passive hyperæmia of the cardiac walls may be said to result only as a part of a more or less general or secondary venous engorgement. Occasionally, however, in cases of acute dilatation, or rapidly established cardiac obstruction, as from the formation of a thrombus, it is not uncommon to find enormous distension of the venous sinuses, and extensive passive congestion of the heart and pericardium. The turgid myocardium has a dark, reddish-brown appearance, and the veins, especially those visible in the sub-epicardial region, are tortuous, large, and engorged. The congested endocardium is slightly darker in colour, less glistening than usual, and the dark and hyperæmic muscle stretches the overlying endothelial lining.

(4) *Hæmorrhage.* — Small localised hæmorrhages into the cardiac tissues are not infrequently found post-mortem. In certain blood diseases, such as scurvy, purpura, infantile scurvy, profound anæmias, especially in the form termed "pernicious," and leucocythæmia, small punctiform hæmorrhages into the pericardium, or scattered through the myocardium, or even sometimes beneath the endocardium, are of common occurrence.

Associated with many of the infectious diseases, especially those of a septic nature, hæmorrhages into the cardiac textures are common. They are also not infrequently met with in certain cases of poisoning, particularly by arsenic and phosphorus.

In states where convulsions have occurred either from toxic influences, as in strychnine poisoning, or from local injury or disease of the nervous structures, small ecchymoses are occasionally found in the tissues of the heart at the autopsy. In conditions, also, where extreme venous engorgement has occurred, as, for instance, in the forms of suffocation, more or less extensive hæmorrhages, particularly into the pericardium, are common. In the final stages of many cardiac conditions small hæmorrhages frequently develop in the walls of the heart as well as in the body generally.

In some cases considerable areas of hæmorrhage may be met with. In an old man with advanced coronary disease we found a large area of hæmorrhage which had occurred into a myomalacic patch, and which, rupturing into the pericardium, led to a fatal issue. Hæmorrhagic infarcts of the myocardium result from lesions of the coronary vessels. Such vascular changes are usually of slow development. Occasionally emboli, particularly in cases of malignant endocarditis, cause acute blocking, with rapid effusion of blood into the adjacent muscles. Aneurysm of the coronary artery may result. The rupture of a cardiac aneurysm is one of the well-recognised causes of hæmopericardium and sudden death.

Hæmorrhages into the cardiac structures are usually situated chiefly in the pericardium or endocardium, or in the tissues immediately beneath. They are generally of very limited extent, and should the patient recover from the condition giving rise to the hæmorrhage, the effused blood is doubtless readily absorbed. Ecchymoses of the pericardium and endocardium must not be confused with post-mortem staining.

Occasionally hæmorrhages into or beneath the endocardium occur in the region of the valves, particularly in connection with infective or malignant endocarditis.

Extensive areas of hæmorrhage into the cardiac substance may be met with in fatal cases of violence. Sometimes, especially in children, much bruising of the heart may occur without rupture into peri- or endocardial cavities.

(5) *Dropsy.*—An œdematous condition of the cardiac textures is always a secondary process. Such occasionally occurs in Bright's disease, and to a less extent in certain hydræmic states of the blood. Not only may there be transudation into the pericardial cavity, but the sub-pericardial tissue, and even the myocardium, often assume a boggy and water-logged appearance. Frequently the surrounding adipose tissue will be found distinctly œdematous. Where large pericardial adhesions exist, the old inflammatory tissue not infrequently presents considerable œdema. Also in the final stages of cardiac disease the heart's tissues participate with the rest of the body in a more or less extensive œdema. This is particularly marked in cases of primary muscle failure. The myocardium, however, even in these cases, from the character of its structure and the nature and arrangement of its fibres, presents but little evidence of extensive serous transudation.

(6) *Thrombosis, Embolism, and Infarction.*—Local clotting may occur either in the cavities of the heart or in the vessels supplying the heart.

The cardiac thrombus may be of acute or chronic formation. It is frequently met with in cases of dilated heart, occurring more particularly in the appendices of the auricles, which are commonly found completely obliterated at the autopsy by firm and often laminated clot. Thrombosis also occurs between the trabeculæ, and sometimes even at the rounded apex of a dilated ventricle.

Thrombosis also constitutes an important process in the formation of extensive vegetations, as occur in such abundance and in so exuberant a form in malignant endocarditis.

By slow deposition of fibrin a thrombus firmly fixed to the endocardial wall may extend forward into the auricular or ventricular cavity, and thus give rise to a "cardiac polypus." When formed in the auricle it may project sufficiently to obstruct the auriculo-ventricular orifice, and give rise to symptoms of valvular disease. They have even been seen as ball-like masses detached from the cardiac surface.

Thrombosis of the coronary arteries is an extremely important condition, cutting off, as it necessarily does, the nutritional supply to the cardiac muscle. It is usually dependent on changes in the vessel walls, arising as a part of a general arterial sclerosis, or sometimes from local atheroma, arteritis obliterans, or simple senile calcification. Occasionally the thrombus is secondary to embolic obstruction, due to detachment of vegetations from the aortic valve or aorta in malignant endocarditis or aortitis.

The endocardium is a birthplace of emboli. It is in the cavities of the heart or on the surfaces of the valves that most of the emboli arise, which presently, we shall have to show, form such important factors in the secondary lesions of cardiac disease.

Cardiac infarction results from obstruction of a coronary artery. Should the occlusion or obliteration of the vessel be associated with extravasation of blood, a so-called "red" or "hæmorrhagic infarct" results. It is important to remember that while embolic blocking of the coronary vessels is comparatively rare, obstruction from sclerosis, atheroma, calcification, and thrombosis is common. In the latter conditions the myocardial areas, rendered anæmic, and becoming the seat of simple softening, constitute a "white" infarct or area of cardiomalacia. The red infarct will present a dark red appearance or mottled aspect, varying from brown to yellow. Sometimes the central parts are white or yellowish-white, and the borders red or brown. After a certain amount of absorption or disintegration of the blood has occurred, the infarcted tissues often assume a yellowish-grey appearance, or in old cases a greyish, translucent, structureless aspect. In some instances the myocardium may present a depressed puckered scar at the seat of the old infarct.

Sometimes the softening, resulting from coronary obstruction, may involve one or more of the papillary muscles, rendering them ineffectual as contractile structures. The softening may also involve the overlying endocardium, and extensive clotting will then occur over the affected areas. The whole depth of the myocardium may even be involved, and rupture occasioned into the pericardial cavity.

The importance of the above processes in the production of fibroid induration of the heart will be referred to later.

When the embolus contains pyogenic organisms, septic myocarditis or an acute abscess of the cardiac walls may result. This rupturing into the pericardium will produce acute septic pericarditis, or, extending into the cavities of the heart, give rise to a general pyæmia.

(7) *Inflammation.*—It is not our purpose here to discuss the nature of the inflammatory process, or the exact relationship of the lesions found to the etiological factors present. Suffice it for the present to indicate the main outlines of the leading characters of such changes as custom and experiment have recognised as inflammatory. The essential features of the inflammatory process may be divided into those of an exudative and those of a proliferative character. These differences would appear to depend, first, upon the nature and intensity of the irritant; and, secondly, on the structure, especially as regards vascularity, of the tissue involved.

The inflammatory process as it occurs in the heart may be limited to either serous, muscular, or connective tissues, although frequently involving all structures. In studying the phenomena of cardiac inflammation due consideration must be given to the anatomical and physiological peculiarities of the affected parts. The exposure of the endocardium to ready infection from irritants in the blood stream; the non-vascular character of the inner lining of the heart and the greater part of the valves; the dependence of the myocardium on the integrity of the blood-supply; the liability of the extension of inflammatory processes to the pericardium and other cardiac tissues; and the influence of the movements of the heart, constitute factors having important bearings on the inflammatory process.

In acute inflammation the exudative factor is usually most marked. The pouring out of coagulable serum, the passage of leucocytes, and the escape of a variable number of red corpuscles, are the chief of the vascular phenomena. At the same time textural changes occur, chiefly of a degenerative character; but in the less acute forms indicating efforts at a reactive process, in which the connective-tissue cells play an important part. Should the inflammatory process affect pericardium or endocardium the products are enabled to escape from a free surface. In the former they collect in

the pericardial sac, but in the latter they may be conveyed by the circulation to all parts of the body, thus readily explaining the general toxæmic manifestations met with in certain forms of endocarditis. In myocarditis the irritant must reach the muscle through the coronary vessels—at present we know but little as to the lymphatics of the heart in pathological states—or else, as often seems the case, by extension from the peri- or endocardium. Here the products of the inflammatory process cannot escape, and either by direct pressure, or the influence of morbid products on the several elements, lead to various degrees of degeneration or actual necrosis. In the less acute and chronic forms of inflammation the proliferative changes become conspicuous. Multiplication of the connective-tissue and endothelial cells leads to the production of tissue which, however, is to be considered as of a decadent type. In the endocardium, and especially in the valves, increased production of tissue, usually of a fibrous character, leads to extensive mechanical defects, as will be indicated in the section on valvular diseases. In the pericardium local or general thickening may result, and frequently the pericardial sac is obliterated by the formation of adhesions. In the myocardium the intermuscular tissue may be increased in amount and rendered of a more fibrous character, and compress or even actually replace the essential muscle elements. Frequently evidences of degeneration and destruction occur in the same case with proliferative changes.

The so-called *infective granulomata* may conveniently be referred to here, closely allied as they are to the process of inflammation as we are accustomed to view it. The specific irritants of tuberculosis, syphilis, and actinomycosis occasionally invade the cardiac tissues. Tuberculous lesions are by no means uncommon in the pericardium. Sometimes large cheesy nodules occur in the subpericardial structures, or even in the myocardium. At times more or less diffuse caseous areas are met with, or a certain degree of generalised fibrosis. Gummata are rare. Exceptionally they develop as caseous-looking yellowish-white nodules, often surrounded by dense fibrous tissue. They may even rupture into the cavities of the heart. Multiple gummata have been found in hereditary syphilis. Syphilis may also give rise to a diffuse interstitial formation of fibrous tissue, and some have claimed that this should be considered an arterio-sclerotic induration rather than a true interstitial myocarditis.

Actinomycosis is characterised by the formation of grey or yellowish-white granulomata which may suppurate, and in which the typical organisms may be found.

(8) *Atrophy.*—This constitutes the simplest form of regressive interstitial change. It may affect the heart as a whole, and be a part of a general atrophy of the body, such as occurs in starvation and diseases like phthisis and diabetes; or it may be local, or at least affect the heart to a greater extent than the rest of the body. Usually the most conspicuous change is in the muscle cells.

Simple atrophy may arise from practically any condition which deranges the normal equilibrium between the evolution and involution of the individual cell. It is of common occurrence in states of premature decay, in all wasting affections, particularly malignant disease and pulmonary tuberculosis, and in affections generally speaking associated with impaired nutrition.

The heart has a small shrunken appearance. The muscle cells are diminished in size, and usually darker in colour from the presence of yellow pigment granules. In advanced forms met with, particularly in senile and cachectic conditions, it may assume a distinctly brown aspect, when the condition is commonly termed "brown atrophy" or "pigmentary atrophy." Non-ferruginous granules become deposited in the muscle cells tending to congregate at the extremities of the nucleus.

(9) *Hypertrophy.*—Cardiac hypertrophy must be viewed as rather a physiological than a pathological process. True hypertrophy of the heart's muscle is always a compensatory measure. It is nature's method of assuring adaptation. It is the reaction to influences making for a disestablishment of cardiac equilibration.

Hypertrophy of the heart frequently arises from the influence of persistent effort, such as is necessarily associated with many forms of laborious work. Such reaction to a physiological over-activity is well seen in blacksmiths and professional runners or rowers. A slight amount of hypertrophy is said to occur normally in pregnancy, possibly as a consequence of increased arterial tension.

Increased activity of the heart from nervous causes may lead to a certain degree of increased muscular development, as is seen in cases of Graves' disease, and so-called functional tachycardia. In most of these instances, however, dilatation seems to precede hypertrophy.

The most marked degrees of hypertrophy are found in conditions of valvular deficiency and states of high arterial tension.

The causes of cardiac hypertrophy may thus be conveniently divided into (i.) intrinsic and (ii.) extrinsic, according as to whether they arise in the heart itself or originate without the heart.

As will be shown when considering the lesions of the different valves, both insufficiency and stenosis may lead to conspicuous hypertrophy. Generally speaking, in the latter form of lesion, since it is usually of slow development and of gradually increasing intensity, and because also the effects of the mechanical defect become chiefly manifest on the walls of the cavity

immediately behind the obstruction during systole, hypertrophy will be the first and earliest secondary change, and probably the most persistent. In valvular incompetence, especially when rapidly established, dilatation is usually the first and most noticeable effect. Hypertrophy, generally speaking, is only a later and often less marked effort at readjustment. Impediment to the free action of the heart may lead to considerable hypertrophy, as is frequently seen in "adherent pericardium": probably here dilatation precedes hypertrophy. Frequently also there are associated endocardial lesions. In conditions of chronic renal disease, particularly in the form of "granular" kidney, very extensive hypertrophy occurs. The increase in muscular development affects chiefly the ventricles, and mainly that of the left.

It may be interesting here to observe that in old age the heart is sometimes found increased in size. Possibly this may be best explained by a reference to the condition of the vessels.

Aneurysm of the aorta is not necessarily associated with any marked degree of hypertrophy. When it leads to any appreciable degree of obstruction which, however, is quite exceptional, or when by involvement of the first part of the arch incompetence of the aortic valve is occasioned, enormous increase in the extent of the walls of the left ventricle may be found.

In pulmonary obstruction, such as occurs in cases of chronic bronchitis, emphysema, fibroid conditions of the lungs, marked hypertrophy of the right ventricle occurs, which, however, is not limited to it, but, as we should expect from a recognition of the solidarity of the heart, is associated with changes in the muscular walls of the left-sided cavities, although to a less extent.

"Idiopathic" hypertrophy is practically an unknown and rightly discarded condition.

Hypertrophy of the heart has been divided into (i.) simple, (ii.) eccentric, and (iii.) concentric. It is doubtful if the last form ever occurs. In clinical and pathological practice the second form, or hypertrophy with dilatation, is usually met with. The so to speak mere mechanical necessities play a determining part. Thus in aortic stenosis almost pure hypertrophy often occurs. In aortic incompetence, since the left ventricle fills from two sources at the same time, a double influence must be considered—one destructive, the other constructive. Hypertrophy, however, can only be maintained when there is adequate nutrition and the expenditure kept well within the cardiac income. This overstepped dilatation becomes the source of cardiac downfall.

It must also be remembered that when hypertrophy is associated with marked dilatation, the larger the cavity the greater the contractile power required to empty it.

True hypertrophy consists in an actual hyperplasia of the individual muscle cells. In many cases, and especially in young subjects, a numerical increase also occurs.

All parts of the heart's muscle share in the hypertrophy, though seldom equally so. The ventricles show the most conspicuous increase in size. The papillary muscles are often greatly hypertrophied. All degrees of hypertrophy are met with. Occasionally the heart doubles both in size and weight.

(10) *Degenerations.*—Impaired nutrition of the cardiac tissues leads to degenerative processes. The normal protoplasm suffers regressive change, and is converted into less highly developed forms. Degeneration of the cardiac structures is always to be looked upon as an involution and strictly pathological. Hard and sharp lines, however, cannot always be drawn between the different varieties, which, in not a few cases, tend to pass from one to the other. Still, generally speaking, and for purposes of description, certain more or less distinct forms may be recognised.

Parenchymatous Degeneration or Cloudy Swelling.—This forms the simplest of regressive changes. It is characterised by a swelling up of the cells and fibres, the protoplasm becoming cloudy, and forming fine free granules with indistinctness or disappearance of the nuclei. The micro-chemical reactions of the granular material show it to be albuminoid. Should the process be arrested at an early stage the affected cells apparently recover. This possibly explains the perfect restoration which occurs in not a few cases of slight degrees of dilatation of the heart.

This form of degeneration is met with particularly in the cardiac muscle cells, and occurs as a result of toxic conditions. It is common in most of the infectious diseases. In typhoid fever, diphtheria, and septic processes the morbid change is usually well marked. It is also met with in acute Bright's disease.

Fatty Degeneration.—This is one of the most frequent pathological conditions affecting the myocardium. It is essentially a different process from fatty infiltration, although the two often occur in combination. It is characterised by a conversion of the cellular contents into fat elements. The change occurs at the expense of the cell protoplasm. It may take place throughout the myocardium, but is more frequently patchy in distribution. The cells have an indistinct granular appearance due to the accumulation of minute fatty globules. In advanced cases the nucleus also disintegrates. Osmic acid stains the fatty granules black, and the cell assumes a vacuolated appearance when the fat is dissolved out by ether.

The macroscopic characters of the tissue differ somewhat according to the extent and duration of the change. The "tabby-cat striation" so

common in pernicious anæmia is due to a patchy distribution which occurs apparently quite irrespective of any alteration in the local vascular supply.

Fatty degeneration occurs in a large number of conditions; in fact, any prolonged morbid state leading to general impairment of nutrition is liable to produce it. It is common in all forms of anæmia, but particularly marked in the "pernicious" variety, and in toxic states, such as from phosphorus, arsenic, or alcohol. It occurs more or less naturally as part of the general disintegration of old age, but here probably at least for the most part in association with a certain degree of actual vascular disease. In pyrexial and toxæmic conditions, especially those associated with the infectious fevers, it is common. Some cases of fatal cardiac syncope in diphtheria are due to this cause. It also occurs in various wasting diseases, and is frequent in cancer, chronic tuberculosis, and all forms of cachexia and inanition. Fatty degeneration may also result from local disturbance of the cardiac nutrition as from long-continued increase of arterial pressure, and especially when this leads to passive venous congestion. The anæmia and atrophy, secondary to coronary obstruction, usually also proceed to fatty conversion of the muscle cells in the affected area.

Hyaline degeneration seems to be dependent on some process akin to coagulation, whereby the cell contents assume a vitreous, shining, translucent appearance. The product formed is a strongly refracting substance and very resistant body, being insoluble in water, staining yellow with iodine, but giving no reaction with chloroform or ether. The fibrous tissue of the vessels is specially affected, but the muscle elements may also manifest the change. The affected textures macroscopically have a distinctly cloudy appearance. As to its exact pathology there is much dispute, but except that it is undoubtedly a manifestation of a regressive metabolism of the cell little can with certainty be said. It occurs particularly in toxæmic conditions. Hyaline and granular degeneration frequently occur in association with other regressive nutritional processes.

Dissociation of the muscle cells, which has been described as "segmentary myocarditis," occurs in several conditions of depraved nutrition, and is sometimes met with in cases in which hyaline and other forms of degeneration occur. Some have contended that this loosened and separated condition of muscle cells is dependent on a post-mortem change.

Mucoid degeneration is occasionally met with in the connective tissues of the heart, occurring perhaps most frequently in the thickened prominences of sclerosed valves. The affected patches have a gelatinous appearance.

(11) *Infiltrations.*—Fundamentally the process of infiltration is quite distinct from that of degeneration, although frequently found in association. Both are to be considered as evidences of impaired nutrition.

Calcareous Infiltration.—Deposits of lime salts are of very common occurrence in connection with the cardio-vascular mechanism. Usually they are met with in the form of carbonates and phosphates, but sometimes in combination with salts of magnesium. All parts of the heart may be involved. Occasionally calcareous plates are found in old pericardial adhesions.

Sclerosed valves commonly present extensive deposition. The coronary vessels are frequently involved, and in some few instances even the myocardium becomes the seat of a plentiful deposition of lime salts.

Calcareous change is particularly liable to affect the valves, and is an important factor in producing and maintaining forms of valvular obstruction. In sclerosed valves large, hard nodular or irregular masses are very common, and go far to render the segments immobile.

Calcareous deposition is said to occur primarily as a manifestation of old age, but it will usually be found to have taken place in structures already the seat of degenerative processes. The true pathogeny of the condition is by no means clear. Apparently local lesions present conditions which predispose to or actually produce a precipitation of the lime salts or a conversion of the soluble into insoluble compounds.

Uratic deposition is said to occur within the cardiac structures in gout, but if such is ever the case it is quite exceptional.

Lardaceous infiltration also affects the heart as a part of a more general involvement. It cannot be considered as of any special clinical importance. The coats of the coronary vessels are earliest affected, but in advanced cases the basement membranes, fibrous elements, and even muscle cells may be affected. As is well known, it occurs particularly in association with chronic syphilis and states accompanied by long-continued suppuration.

Fatty infiltration frequently occurs in combination with fatty degeneration. As already indicated, they are, however, essentially different processes, the former characterised by a mere cellular deposition, the latter arising from actual protoplasmic conversion.

Fatty infiltration or cardiac lipomatosis is commonly but a part of a general obesity. Deposition of fat occurs particularly in the pericardium, the subepicardial tissue, in and along the septum, and around the base of the heart. The whole organ may be enclosed in a panniculus adiposus. Where excessive the cardiac action may be much impeded, and atrophy and degeneration of the muscle cells not infrequently result. Rupture of a fatty heart has occurred spontaneously from a comparatively trifling cause.

This condition of fatty infiltration arises either from increase of intake or diminution in expenditure. It thus occurs in large eaters, chronic alcoholics, or in states where, as in some forms of anæmia, there is diminished oxidation.

Certain families, and women particularly in advanced middle life, are peculiarly prone to develop this condition.

(12) *Necrosis* forms the final and most serious form of the degradation processes. Actual death of cardiac tissue may occur in several forms:—

Simple softening, constituting cardiomalacia, occurs as the result of obstruction of a coronary vessel from a mechanical or non-septic cause. When a large branch is involved, and consequently a wide area of muscle rendered anæmic and subsequently necrotic, death may occur from rapidly established cardiac failure. In other cases the softened area gives way, and allows of rupture of the cardiac wall, with escape of blood into the pericardium. When the softened area is absorbed and replaced by scar tissue, a cardiac aneurysm may result.

Ulceration or molecular necrosis constitutes a very important feature of malignant endocarditis. Here the microbial invasion, or the toxins resulting from their growth, produces extensive death of the structures forming the affected valve or mural endocardium. The necrotic tissue becomes entangled with blood and fibrin, increasing the production of the abundant vegetations so characteristic of this form of endocarditis; softens into minute granules, which are washed away in the blood-stream; or, being detached in fragments of varying size, is carried along and lodged in some distant vessel as emboli. Such accidents, however, are not limited to ulcerative endocarditis, as will be shown presently. Sometimes in septic endocarditis the valvular tissue yields, so producing an acute aneurysm of the valve.

Occasionally necrotic foci are met with in the myocardium due to the penetration of organisms from the endocardium or their conveyance by means of the coronary vessels. An embolic plug, proceeding from vegetations of a malignant type, may lead by a process of rapid softening to dilatation of a coronary artery and the formation of an acute aneurysm.

Gummatous and caseous necrosis are only very exceptionally met with in the substance of the heart.

(13) *Tumours.*—Primary growths of the heart are very rare, and little more than pathological curiosities. Sarcoma, fibroma, myxoma, and myoma have all been described. Some are said to be of congenital origin. Occasionally they assume a polypoid form, and extending into the cavities of the heart may produce cyanosis, and give rise to evidences of cardiac obstruction and muscular deficiency.

Secondary growths are more frequent. They may either be of a carcinomatous or sarcomatous type. Melanotic sarcomata occur sometimes as numerous and extensive secondary deposits in the myocardium. Secondary nodules of growth may be found embedded in the walls of the heart or projecting from its inner and outer surfaces. Sometimes sarcomata extend directly to the pericardium and heart from the mediastinum. Cancer of the œsophagus very exceptionally extends to the heart.

(14) *Parasites* of the heart are very rare in this country. The cysticercus form of the *Tænia solium* is occasionally met with, and is said to occur most frequently in the walls of the left ventricle. It varies in size from a pea to a marble. Hydatids, or the cystic form of *Tænia echinococcus*, may develop into cysts of considerable size. When rupture occurs into the cardiac cavities embolism of the pulmonary or systemic vessels may result.

These parasitic involvements of the heart are of but little clinical importance, and do not call for further consideration here.

III. Effects of Cardiac Disease

Many of the secondary results of cardiac disease are chiefly of importance from their clinical significance, and are best dealt with from that standpoint. For the present purpose it will be sufficient to indicate only the leading pathological features of practical interest.

Passive venous congestion is one of the most important of the consequences of cardiac failure. Its essential character is blood stagnation.

Cyanosis.—Every venously congested tissue tends to assume a deep red, livid, or even bluish tint. The condition arises from any cause impeding due aeration of the blood. In many forms of congenital defect of the heart it is a specially conspicuous feature (morbus cæruleus). In more or less all cases of cardiac failure, when the right cavities become distended cyanosis appears, and deepens in intensity with the progressing embarrassment of the pulmonary circulation. Cyanosis is especially marked in many cases of mitral stenosis. It is also a conspicuous feature in most cases of primary muscle failure.

The cyanosed tissues vary in colour from a slight dusky red to a deep purple-black. The most intense degrees are usually best seen in peripheral parts of the circulation.

Passive Hyperæmia of the Lungs.—Passive pulmonary congestion is sometimes associated with pulmonary œdema. Venous engorgement may, however, be an early result of cardiac disease and long maintained as almost the only serious effect. It is met with in its most marked form in mitral stenosis, in which the pulmonary tissues may be subjected to passive hyperæmia for years before the failure of the right ventricle allows establishment of a general venous engorgement. This pulmonary congestion is often in cases of mitral disease

relieved from time to time by hæmoptysis, which may prove a natural and beneficial venesection in the subjects of mitral stenosis.

As the result of long-continued passive congestion the lungs pass into conditions of so-called "red" and "brown induration." A cyanotic lung is of increased consistency, its elasticity diminished, and generally increased in weight, though often lessened in size. In colour it varies from a red to a deep brown, according to the degree and duration of the engorgement. The connective tissue is usually increased and the air vesicles diminished in size, while their walls are thickened. The capillaries are much distended, tortuous, and hæmorrhages readily occur.

"*Nutmeg*" *Liver*.—The hepatic tissue is one of the first to fall under the influences making for passive hyperæmia. Failure of the heart to empty its right-sided cavities necessarily interferes with the return of the blood from the inferior vena cava. Passive hyperæmia of the hepatic veins ensues, and if prolonged leads to such changes in the appearance of the liver substance as is aptly characterised by the terms "myristicated" or "nutmeg." The organ is often enormously enlarged and much increased in weight. In states of venous congestion the sponge-like adaptability of the organ becomes of considerable practical importance. The liver substance is dark red in colour, and, as seen through the smooth and stretched capsule, has a mottled appearance. On section the large veins are found distended, but after death the alteration in blood-pressure allows the thin-walled vessels to collapse and the liver to shrink to a considerable extent, so that frequently the pathologist finds an organ which does not correspond in size to that mapped out during life.

The cut section of a "nutmeg" liver presents a mottled appearance; the central part of each lobule is of a dark reddish-brown colour, due to distension of the intra-lobular vein and engorgement of the capillaries, with atrophy of the adjacent liver cells. The peripheral zone of the lobule is of a grey or yellow colour, due to extensive fatty changes in the liver cells, and in advanced cases the atrophy, degeneration, and pigmentation with bile may be extensive.

"*Cyanotic*" *Spleen*.—The spleen participates in the results of obstruction to the portal blood-flow, hence, in most cases where the effects of impeded blood-flow from cardiac disease are transmitted to the liver, the spleen sooner or later participates in the portal venous engorgement. It is interesting to note, however, that the capillaries of the liver may long successfully act the part of a protective buffer to the portal system. A cyanotic spleen is of a dark purplish-red colour, firm in consistency, tough, and not readily friable. It is generally enlarged to some extent, but not always so. In cases of congestion from hepatic cirrhosis the spleen is usually much larger and heavier than when of "cardiac" origin.

Passive congestion of the gastro-intestinal tract is usually well marked in cases dying from progressive heart failure. The veins of the stomach and intestines are engorged, the mucous membrane swollen, and frequently small hæmorrhages occur. It is important to note that cyanotic conditions of these structures readily pass into a state of actual inflammation. It is quite common to meet with evidences of gastro-intestinal catarrh in fatal cases. Hæmorrhoids and sometimes œsophageal varices may be present.

Passive venous congestion of the pancreatic and other branches of the portal vein lead to impairment of the function of the organ concerned, and the engorgement of the peritoneum leads to the establishment of ascites; and in long-standing cases chronic indurative processes seriously damage the absorptive properties of the peritoneum. Special note should be made of the porcelain-like thickening of the capsules of the liver and spleen, and other parts of the serous membrane of the abdomen met with in long-standing cases of ascites of cardiac origin.

Cyanotic kidneys are met with in all cases of long-continued heart disease. The organs are enlarged, increased in weight, of firm consistency, having a dark brownish-red colour and with prominent stellate veins. The capsule may not separate very readily, but the surface is generally moderately smooth. Occasionally old scar-like depressions are met with. Both cortex and medulla are increased in extent, of a dull red or purplish colour, with the venæ rectæ and glomeruli often prominent. Microscopic examination shows great distension of the veins and usually distinct changes, mainly degenerative, in the renal epithelium. In many instances it is clear that no hard and sharp line can be made, either pathologically or clinically, between states of venous stasis and actual inflammation. It is certain that in the kidney conditions of the former readily merge into those of the latter. In cyanotic kidneys minute hæmorrhages are frequently found, as also pigment granules in the cells, and not uncommonly hyaline casts in the tubules.

Congestion of the genital organs, when occurring in the female, may be of importance in explaining the occurrence of menorrhagia or metrorrhagia.

Passive hyperæmia of the superior vena cava and its branches is manifested by engorgement, and sometimes by tortuosity of the veins of the neck and face. Special reference may here be made to certain curious cases of so-called "alcoholic" heart, which clinically much resemble cases of mediastinal tumour at the first glance—the venous engorgement and dropsy of the head and neck and upper part of the trunk and upper extremities being often extreme.

The brain and its membranes are also involved in the venous distension, and this explains many of the troublesome cerebral manifestations met with in cardiac cases.

Local anæmia occasionally occurs as a seeming consequence, or, at least, accompaniment of cardiac disease. In malignant endocarditis general anæmia of rapid production may be very marked. Mitral stenosis is sometimes found in young chlorotic females. Cardiac dilatation is by no means exceptional in simple anæmia. A fatty and anæmic heart is sometimes the only explanation of a fatal syncope.

Dropsy forms one of the most important consequences of cardiac failure. Serous transudation may occur into the subcutaneous tissues constituting anasarca, into the texture of organs as in pulmonary œdema, or into serous cavities forming hydrothorax, hydroperitoneum, and ascites. Several factors play a part in its production. Mechanical obstruction to the return of venous blood, alteration in the walls of the blood-channels, depraved conditions of the blood and derangement of the lymphatic circulation, may all be of importance. The first, however, forms the most evident explanation. Impediment to the return of venous blood is, as already indicated, the most constant and striking feature in cardiac failure. Generally speaking, when dropsy occurs it is more or less proportional to the degree of venous engorgement. This, however, is not always so. In many cases very advanced degrees of cardiac disease may be attained without any sign of dropsy, although it may appear almost suddenly shortly before the end. Hence, although there is some evidence in favour of the contention that no direct relationship exists between the amount of heart disease and dropsy, it must be insisted upon that usually dropsy develops with the failure of the vigour of the cardiac muscle, and in most instances is essentially proportional to and progresses with the muscular embarrassment.

It will be shown elsewhere how readily a heart in unstable equilibrium may be influenced by comparatively trifling agencies, and the adjustments of compensation which depend on muscular adaptiveness may be suddenly upset, so allowing of the rapid development of dropsy. A readjustment may be followed by as rapid a disappearance of the transuded fluid. The importance of such predisposing influences as a severe accident, mental shock, acute disease, and the like, cannot be over-estimated in clinical work. Mechanical conditions, which in health would have no appreciable influence, may in cardiac derangement prove sufficient to determine the occurrence and regulate the distribution of serous transudation. The common occurrence of œdema of the feet and legs in early or slight forms of cardiac asthenia is sufficient proof of this.

But a merely mechanical view is far too limited to explain all the causation of dropsy in cardiac disease. Disturbance of the nervous mechanism, altered nutrition of the walls of vessels, impoverished conditions of blood, will lead to transudation, and in some heart affections there is reason to believe that some distinct derangement of a peripheral mechanism may be the true explanation. It is only by admitting such a view that one can explain the curious localisation of œdema—as, for example, that over the sternum and sacrum, or even into the scalp, such as is by no means exceptional in cases of alcoholic muscle failure.

Comparatively little, however, is known as to the local conditions making for an increased permeability in cardiac conditions. In long-continued states of vascular derangement nutritional changes must of necessity occur in the vessel walls, and probably do much to maintain tendencies to transudation.

It must also be remembered that in most cases of cardiac failure there are influences greatly impairing the normal lymphatic circulation, since the integrity of the latter is necessarily in great part dependent on the perfect adaptability of arterial and venous blood-pressure.

It must also be admitted that anæmia may be a factor in the subjects of ordinary valvular heart disease, so to speak, promoting œdema.

The dropsical effusion is usually a transparent yellow or yellowish green fluid, with few or no cellular elements, showing no tendency to spontaneous coagulation, of low specific gravity and containing but little albumin or other solids. The fluid, however, probably varies in character, as would seem to be sufficiently shown by the free running or otherwise through a Southey's trochar.

Œdema of the lungs is of special importance in heart disease. Usually it develops in organs the nutrition of which has been long impaired. Occasionally it arises with startling rapidity. The lungs are large and bulky, much increased in size and weight, pale, and pit on pressure. On section clear, frothy, serous fluid escapes, often in enormous quantities.

Thrombosis, Embolism, and Infarction.—Thrombosis may occur as a consequence of endocarditis and morbid changes in the lining of the heart and vessels. It is common in dilatation of the cardiac cavities, and also arises secondary to embolic obstruction. Reference has already been made to the occurrence of thrombosis in the cardiac cavities and coronary vessels. Local clotting also sometimes occurs in heart cases in the peripheral vessels when the propulsive force of the organ has suffered much diminution. Occasionally local œdema of an extremity, developing as a terminal condition, is found to have been due to a thrombus. Local coagulation may affect either the arteries or the

veins; when of embolic origin it is of course always arterial. The medium-sized vessels seem to be most prone to be involved.

The embolus occurring in cardiac disease is commonly formed by blood-clot, but sometimes it may arise from a detached fragment of a calcified valve, a portion of vegetations, or parts of a softening thrombus. In endocarditis embolism is common. It also frequently occurs in cases of dilated heart, and particularly in such cases of valvular disease as mitral stenosis, where cardiac thrombi are especially liable to form between the musculi pectinati of the left auricle, in the left auricular appendix, and around and between the columnæ carneæ of the left ventricle. The manifestations of embolism vary according as to whether the obstructed vessel is furnished with a free anastomosis or is terminal. In the former the effects are usually temporary and comparatively trivial. Obstruction of so-called end arteries, such as the pulmonary, renal, splenic, and coronary arteries, the central artery of the retina and the nutrient arteries of the brain, to which may also be added for practical purposes the superior mesenteric artery, leads to results more or less serious, and frequently producing permanent defect. The consequences of such blocking are engorgement, hæmorrhage, and necrosis occurring separately or in combination.

Acute aneurysms may arise in the cerebral and coronary vessels as a result of embolic blocking in septic endocarditis.

Hæmorrhagic infarction of the lungs is an almost constant accident of severe cardiac disease. The affected portion, varying in size from a minute patch to a mass involving the greater part of a lobe, is rendered solid, of a deep red colour, and often wedge-shaped. The lung alveoli are distended with blood, and the adjacent vessels engorged. Frequently an "embolic" pneumonia in the neighbourhood of the infarct greatly extends the area of consolidation. Some pathologists claim that many of the pulmonary infarcts are not of embolic origin, but are to be looked upon as true hæmorrhages.

Renal infarcts are common in heart cases. They are usually of the so-called white variety, presenting yellowish-white wedge-shaped areas with the base at the surface of the cortex, and generally with a surrounding zone of hyperæmia.

Splenic infarcts often assume considerable dimensions. Various gradations between the white and the red occur. Commonly they form large wedge-shaped but sometimes irregular yellowish-white areas surrounded by a zone of congestion.

Obstruction of the cerebral vessels leading to infarction of the brain, as it may be considered, usually produces a simple white softening or encephalomalacia; but in some cases, as for example in malignant endocarditis, there may be red softening, or even such copious hæmorrhage as to obliterate all evidence of the producing embolus.

Infarction of the myocardium may itself occur from embolism of the coronary vessels. It leads to a condition of cardiomalacia, but occasionally a distinct "hæmorrhagic infarct" is produced.

In heart cases it is by no means uncommon for the pathologist to discover depressed, puckered cicatrices and remnants of scar tissue in the spleen, kidneys, and lungs, evidencing the occurrence of previous infarction.

Reference may here be made to the fact that in most cases of malignant endocarditis the resulting infarcts are usually simple, presenting no tendency to undergo suppuration, although in some of the pyæmic varieties distinct abscesses result.

Hæmorrhages are of common occurrence in heart disease. Usually they are associated with states of passive hyperæmia, embolism, or infarction.

Numerous minute cutaneous hæmorrhages are frequently met with in the final stages of cardiac affection, being particularly common about the wrists and back of the hands, but especially met with in the lower extremities.

In malignant endocarditis hæmorrhages into the skin and mucous membranes are of common occurrence.

Such forms of hæmorrhage as epistaxis, hæmoptysis, hæmatemesis, apoplexy, hæmaturia, and metrorrhagia will be best dealt with clinically.

Altered Blood States.—From what has been said regarding the secondary anæmia and hyperæmia occurring in heart disease it will be manifest that profound changes must occur in the blood.

In malignant endocarditis leucocytosis is usually well marked, and may prove of diagnostic service.

In cyanosis the blood is visibly darker than normal. Its density is increased. The hæmoglobin is raised in amount, and both forms of corpuscles increased. This increase is said to occur in congenital as well as acquired forms, and in both primary and secondary cardiac failure. Such changes are apparently not to be explained by a mere concentration of the blood arising from transudation of its more watery parts, neither are they altogether compensatory to the cyanosis, but are due probably, as Gibson suggests, to the fact that the functions of the corpuscles being lessened the wear and tear which they undergo is reduced, and the duration of their individual existence prolonged. It is, however, not clear if the condition of the blood in the superficial and more accessible parts may not vary from that in the deeper regions of the body.

Jaundice is a common condition in advanced stages of heart disease. It is generally indica-

tive of the degree of engorgement of the liver, but may arise from an intercurrent attack of gastro-intestinal catarrh, which is by no means rare in cardiac affections. In some cases of malignant endocarditis well-marked icterus appears, and very intense jaundice is often seen towards the end of mitral cases.

Toxæmia occurs in the final stage of some few heart cases, particularly malignant endocarditis; but in the greater number of forms of cardiac lesion there is not much evidence of toxic involvement unless CO_2 be included.

Nutritional changes are specially noticeable in congenital heart disease, or lesions established early in life, "clubbed fingers" particularly becoming very marked. In a few cases of aortic incompetence failure of nutrition occurs for which there is no obvious cause.

Rupture of the heart, although sometimes described as "spontaneous," is probably in most if not in all cases to be looked upon as a terminal result of antecedent disease. Usually lesions of the coronary vessels are present, but these and the changes observed in the myocardium are referred to elsewhere.

Sudden death is one of the most striking of the pathological and clinical effects of cardiac disease. It is, however, not usually so frequent as is generally considered to be the case. It is rare for death to occur without some evidence of cardiac embarrassment having been present. In not a few cases, however, the final stages may be rapidly passed through. More usually cardiac failure is progressive, and takes time for its evolution. In valvular affections sudden death is peculiarly associated with aortic incompetence. We have also met with several instances of sudden cardiac failure in aortic stenosis, a lesion not usually supposed to lead to such a termination. In mitral affections death is generally slow. In some cases of muscle failure sudden death is by no means infrequent. Degenerate conditions of the myocardium, as in the notorious "fatty heart," and disease of the coronary arteries also frequently lead to a like conclusion, in the latter case angina pectoris supervening.

A consideration of the intrinsic effects of cardiac lesions, such as hypertrophy, dilatation, and other changes involving the myocardium and endocardium, may be best left until the more important individual lesions are described. The mechanism of compensation will be more clearly understood after the various special valvular lesions have been dealt with.

B. SPECIAL PATHOLOGY

Having now completed our general survey of the etiological factors of heart disease, and considered the morbid processes and their effects, it becomes necessary to describe the individual lesions met with in the more important cardiac affections.

1. Endocarditis

Definition.—An inflammatory process involving the internal or lining membrane of the heart, and especially the valves.

General Etiology.—Considerable uncertainty exists as to the relative importance to be attached to the different causal factors, and hence a perfectly satisfactory etiological grouping is not at present available. Manifestly the irritant must in all cases reach the endocardium by means of the blood. In most forms of endocarditis micro-organisms have been discovered, and many pathologists claim that all varieties are of microbic origin. The so-called "malignant" cases are undoubtedly due to parasitic invasion, and hence to them the term "infective" is strictly appropriate. The exact cause, however, of the "simple" or "benign" forms, such as are more particularly associated with acute rheumatism, still remains obscure, and is likely to continue so until the true nature of the rheumatic excitant is demonstrated. Some pathologists hold that while "malignant" or "infective" endocarditis is due to virulent micro-organisms, "simple" or "rheumatic" endocarditis results from the action of attenuated forms. However this may be, certainly both pathological and clinical gradations can be traced in the forms of endocarditis, and there is something in favour of the view that the varying lesions really represent different degrees of intensity of essentially the same process. But it is quite possible that certain toxins, including the more or less hypothetical "rheumatic" toxin, are capable of initiating the morbid condition. It will thus be manifest how desirable it is at present to lay aside hard and sharp definitions, and to use our available groupings mainly as means whereby a more intimate knowledge of the different etiological factors may be attained, and a more perfect classification rendered possible.

Classification.—It has long been customary to distinguish (1) simple or benign from (2) malignant endocarditis, and on clinical as well as pathological grounds such division may well be retained, and in accordance with the same we shall consider the two varieties separately.

Other classifications are also adopted:—1. According to the severity and duration—(i.) acute, (ii.) subacute, and (iii.) chronic. 2. Dependent upon localisation—(i.) valvular and (ii.) mural. 3. When the lesion appears originally strictly local, and there is no evidence of any channel of infection, it is conveniently spoken of as—(i.) primary or protopathic, while in the majority of cases it is manifestly (ii.) secondary or deuteropathic. 4. According to the character of the lesion cases are divided into (i.) warty or verrucose, (ii.) polypous or villous, (iii.) ulcerative, and (iv.) suppurative. Combined forms are also frequent. 5. Judged by

the tendencies and consequences of the morbid process, the endocardites have been divided into (i.) necrotic or destructive; (ii.) proliferative, formative, or sclerosing; and (iii.) adhesive.

Simple Endocarditis

Syn. — Benign, rheumatic, papillary endocarditis.

Simple endocarditis is met with in varying degrees of severity, and (i.) acute, (ii.) subacute, and (iii.) chronic varieties are usually described. A consideration of the chronic form may be best deferred until valvular diseases are dealt with. The acute simple variety alone need be mentioned here.

Etiology.—At present it is impossible to clearly distinguish between exciting and merely predisposing influences. While several causes are usually suggested, one causal agent stands out as pre-eminent—rheumatism. Many consider it as sometimes a primary rheumatic manifestation. Certainly endocarditis occurs in association with acute polyarticular rheumatism, or "rheumatic fever," in proportions variously estimated as from 20 to 80 per cent. The younger the subject the more common the association. It is sometimes said that the more violent the articular rheumatism, the greater the coincidence of endocarditis, but in a large number of cases it develops in association with very mild or even unrecognised attacks of rheumatism. The first attack of rheumatic fever usually proves more damaging than later ones. Usually the endocarditis commences early in the course of the rheumatic attack, although the condition may be undetected at the time.

Sometimes endocarditis develops during or follows less manifest rheumatic processes. The endocarditis occurring in scarlatina is sometimes, and probably in gonorrhœal rheumatism always, of an "infective" type. Endocarditis is rare in monarticular rheumatism and in chronic rheumatism.

Amongst 29 cases submitted to autopsy with apparently acute "simple" endocarditis, in the Pathological Department of the Manchester Royal Infirmary, a clear history of rheumatism had been obtained in 17, and there was reason to believe that rheumatic manifestations had been present in even a larger number. In 20 there was also evidence of old-standing valvular disease.

Whether or not chorea is a manifestation of rheumatism as some would believe, it is at all events commonly associated with rheumatism and endocarditis. Probably some degree of endocarditis occurs in from 40 to 60 per cent of all cases of chorea.

Endocarditis occasionally occurs in connection with the infectious fevers, particularly scarlet fever. Quite exceptionally endocarditis arises in the course of other acute infectious diseases, such as measles, small-pox, diphtheria, typhoid, and malaria, and is possibly then truly of "infective" type.

It is also said that acute endocarditis has in some few instances followed injury to the chest or rupture of a valve by strain.

Among the predisposing conditions reference may be made to the following:—In young subjects acute endocarditis occurs with somewhat greater frequency in females. In later life the difference of sex is less marked. It is most common in young subjects and in early adult life. It is exceedingly rare in old people, a first attack being then practically unknown. The average age of fatal Manchester cases of acute endocarditis was a little over 26 years. Pre-natal endocarditis undoubtedly occurs; indeed some pathologists consider fœtal endocarditis as by no means rare. Endocarditis is not infrequently associated with malformations, particularly of the valves.

Transmitted influences are of importance, apparently only in so far as they lead to the development of rheumatism.

Conditions of climate, social position, occupation and habit, are probably only of moment as they expose the subject to the influence of the exciting agents of endocarditis, particularly rheumatism.

Morbid Anatomy.—The lesions met with in acute endocarditis are usually most conspicuous in connection with the valves, and principally affect those of the left side of the heart, except in fœtal life. The mitral valve is most commonly involved, but in fatal cases vegetations are usually found on both aortic and mitral segments. The vegetations usually appear as a row or fringe of bead-like prominences on the auricular aspect of the auriculo-ventricular cusps, and on the ventricular aspect of the sigmoid segments, but situated a little distance from the free edge along a line corresponding to the points of maximum contact. Sometimes the vegetations are large and assume pedunculated or villous-like shapes. The development of such large fungating masses is a more characteristic feature of malignant endocarditis. Sometimes a few vegetations will be found on the chordæ tendineæ, but in the simple form of endocarditis they do not generally extend to the mural endocardium.

In fatal cases care must be taken to carefully distinguish between (i.) the lesions depending on the endocarditis, and (ii.) those merely associated with the endocarditis.

Acute pericarditis is common. Inflammatory and degenerative myocardial changes are frequent, and probably account for the rapid dilatation which occasionally leads to a fatal issue. Infarction is much less common than in the malignant form. Adherent pericardium is not infrequently met with in fatal cases, generally showing that the fatal attack has been preceded by former similar attacks.

The effects of simple endocarditis may be

briefly considered. They may be (i.) local, and (ii.) general.

Resolution with absorption of the inflammatory products and more or less complete repair of the affected portion of the endocardium very probably occurs in a number of the milder cases.

Adhesion, cicatrisation, and induration result in a considerable number, leading to persistent valvular disease.

Aneurysms of the valves are occasionally formed, but are much more likely to occur in "infective" endocarditis.

Rupture of a valve or chordæ tendineæ is only of exceptional occurrence.

Local myocarditis and fibrosis of the musculi papillares form far-reaching consequences.

Secondary infection leading to the development of a "malignant" endocarditis is a possibility ever to be borne in mind.

Anæmia when it occurs is probably due to the influence of the rheumatic "toxin." It possibly predisposes to the development of infective endocarditis.

Embolism is comparatively rare in simple endocarditis, while in infective endocarditis, and in chronic valvular disease, it is of frequent occurrence.

Morbid Histology.—The minute changes consist in a proliferation and desquamation of the endothelial cells; multiplication of the connective-tissue cells of the subendocardial layer; infiltration with leucocytes, serum, and fibrin; and deposition of fibrin and blood-clot on the free surface from the circulating blood. More or less localised myocarditis is apt to occur, with inflammatory infiltration of the inter-muscular tissue and lymph spaces.

Micro-organisms are sometimes found in the superficial parts of the vegetations, but usually not in the deeper structures. Many hold that this is evidence that the organisms are not to be considered as etiological agents, but rather dependent on a "terminal" infection.

Subacute Endocarditis

This form scarcely calls for special description. Both simple and malignant forms may run a subacute course. Associated with the proliferative process are more or less well-marked evidences of conservative efforts characterised usually by the formation of fibrous tissue and resulting in a certain degree of deformity of the affected parts. Adhesions between the cusps and chordæ tendineæ are liable to occur. The condition readily passes into a chronic stage producing conspicuous valvular deficiencies.

Malignant Endocarditis

Syn.—Arterial pyæmia, infective, mycotic, septic or ulcerative endocarditis.

Definition.—A process resulting from the invasion of the endocardium by micro-organisms, and characterised by proliferative, destructive, or suppurative changes.

Etiology.—In pre-bacillary days this form of endocarditis was recognised, but it remained for the bacteriological investigations of recent years to clearly demonstrate its dependence on microbic infection.

Although always the outcome of microbia invasion, the process cannot, however, be considered a distinct pathological entity, for it probably results from infection by several distinct species of organisms.

In some instances the involvement of the endocardium appears as (i.) a primary lesion; but in perhaps the majority it is found to be (ii.) secondary, either to some other infective process occurring within the body, or having some channel of introduction whence the infective organisms can be definitely traced, as from wounds or septic foci.

Thus three classes may roughly be distinguished:—

i. Where there is no demonstrable source of infection or evidence as to the channel of microbial entrance.
ii. Where there is an evident source of infection.
iii. Where the process is associated with other infections, as some of the infectious fevers, pneumonia, diphtheria, typhoid, tuberculosis, and the like.

The organisms most frequently met with (i.) belong to the pyogenic group, and include:—

Streptococcus pyogenes (including the streptococcus of erysipelas).
Staphylococcus pyogenes albus.
Staphylococcus pyogenes aureus.
Bacillus pyogenes fœtidus.
Bacillus coli communis.

ii. Organisms occurring in connection with other diseases occasionally play an important part, and the following have been detected in the vegetations and ulcerations:—

Diplococcus pneumoniæ.
Pneumobacillus of Friedländer.
Tubercle bacillus.
Typhoid bacillus.
Bacillus of diphtheria.
Bacillus of influenza.
Gonococcus.

iii. Some few organisms not as yet known to be associated with any other infective process have also been described:—

Bacillus endocarditis griseus.
Micrococcus endocarditis rugatus.
Micrococcus zymogenes.
Bacillus endocarditis capsulatus.
Bacillus immobiles et fœtidus,

and a number of others with less defined special characteristics.

It is certainly at present difficult, if not impossible, to apportion the relative importance of the different organisms in initiating and maintaining the morbid process. In some instances the condition is apparently the direct result of some definite microbe, and where such can be proved to be the case, the term "infective" should be replaced by that of the specific agent, *i.e.* streptococcal endocarditis, gonococcal endocarditis.

In not a few cases, however, the condition would appear to be the outcome of a mixed infection; at all events, either by later, or terminal, or post-mortem invasion, more than one form of microbe is present. But this is only in accordance with what is known to occur elsewhere in connection with infective processes.

Unfortunately, examination of the blood during life does not usually afford positive results.

The experimental establishment of endocarditis need not be dwelt on here; suffice it to say that investigations on the lower animals —rabbits and dogs—have clearly demonstrated the possibility of developing a condition practically identical with the malignant endocarditis of man.

But there are important predisposing influences. Malignant endocarditis is commonly found to be engrafted on a chronic inflammatory or degenerative condition of the valves. Sometimes a malformation, possibly due to fœtal endocarditis, has been noted. In some few instances an imperfect ventricular septum has been found. In an analysis of 65 fatal cases of malignant endocarditis occurring in the Manchester Royal Infirmary, of whom not quite 34 per cent gave a clear history of rheumatism, in no less than 50 or 77 per cent was there distinct evidence of old endocardial mischief. In 2 there was adherent pericardium; mitral stenosis existed in 9; aortic stenosis in 2. In one there was congenital malformation. In another the aorta was narrowed and thickened. In several the affected valves were also the seat of calcareous deposition. In only 12 of the cases were there fairly reliable grounds for believing that the valves had been normal previous to the development of the malignant endocarditis.

Occasionally in the course of acute polyarticular rheumatism malignant endocarditis develops. Usually, however, rheumatic fever gives rise to the simple or benign form, and it is quite possible that the "rheumatic" pains complained of in some infective cases should be looked upon as of a septic nature.

Chorea also, although quite exceptionally, is followed by malignant endocarditis.

As already indicated, the organisms of not a few of the general infectious diseases are capable, under certain circumstances, of acting as special pathogenic excitants to the cardiac valves. Chief amongst these may be enumerated pyæmia, erysipelas, pneumonia, typhoid, diphtheria, scarlet fever, influenza, dysentery, and malaria.

Malignant endocarditis sometimes follows parturition, when it may be considered as secondary to a puerperal septic infection.

Suppurative conditions may lead to this form of cardiac involvement. Pyogenic organisms not infrequently gain access to the blood-stream from a local collection of pus or from suppurating wounds. Thus such conditions as empyema, local purulent peritonitis, otitis media, suppurative meningitis, suppurative cholecystitis, have been followed by malignant endocarditis.

It must not be forgotten that when the case comes under observation, no trace of the channel of infection may be detected, and the patient may make no mention of one ever having existed.

Traumatism in the form of injuries to the external surfaces of the body, or internal surface membranes, may become of etiological importance, in so far as thereby channels for the invasion of organisms are established. There is also reason to believe that blows on the chest occasionally lead to the development of an infective endocarditis, possibly by producing some minute injury to the valve segments or endocardium, and so affording access to organisms which may be present in the blood.

Season and climate have been thought to be of some moment. It has been suggested that malignant endocarditis occurs most frequently in the autumn months, but this is very doubtful. In some parts of the country it seems to be much more frequently met with than in others. Some have attempted to show that the affection was connected with decaying vegetation. Malaria probably does not do more than predispose. It is reasonable to believe that all conditions leading to deterioration of health should be credited as being more or less influential as predisposing factors.

Men are considerably more liable than women. Of 65 fatal Manchester cases 46 were men, 15 women, 3 boys, and 1 girl; that is, 49 males and 16 females, or a percentage of a little over 75 for men and nearly 25 for women.

Malignant endocarditis is essentially a disease of adult life. The large majority of cases are met with between the ages of 20 and 40. In the cases referred to the average age was $30\frac{1}{2}$ years. The youngest was 6; the oldest 72 years.

Morbid Anatomy.—The extent and character of the lesions met with in the heart vary considerably. An attempt has been made to recognise varieties according to the predominant feature, (i.) vegetative, (ii.) ulcerative, and (iii.) suppurative, but such a division does little more

than indicate stages, and all conditions may be met with in the same case.

The valves are more particularly affected, but the chordæ tendineæ and parietal endocardium are frequently involved, and the process sometimes extends into the aorta. The left side of the heart is generally affected, although the tricuspid and pulmonary valves may also be involved.

Of the 65 Manchester cases, in only one instance was the malignant endocarditis limited to the right side. In 5 both right and left cavities were involved, the tricuspid and mitral in 2, and the tricuspid, mitral, and aortic in 2. In only one case were the pulmonary valves affected, and their vegetations were found in the pulmonary artery and right ventricle. In 17 the mitral segments were alone involved, in 18 the aortic. In 20 both aortic and mitral valves were the seat of vegetations. In a considerable number the process had extended from the valves and chordæ tendineæ to the adjacent parts. In several the wall of the left auricle was involved. In 4 cases vegetations were present in the aorta itself. In one they extended into the coronary artery. In another they surrounded the opening of one of the coronary vessels. As to the character of the lesions, in 25 there was extensive formation of vegetation, in 34 more or less ulceration. In one there was local suppuration, and in another the mitral valve was the seat of an acute aneurysm. In at least one instance there was apparently recent calcareous deposition in the vegetations.

The vegetations vary greatly in size and extent. Sometimes they are small and scanty, frequently they form large, irregular, pedunculated, shaggy masses. They are generally of a grey or pinkish-yellow colour, and often covered with blood-clot. Sometimes, as already indicated, they are the seat of a recent calcareous infiltration.

The destructive character of the process is commonly indicated by the presence of ulceration. Sometimes the ulcers are small and superficial; more frequently they are large and deep, penetrating through the valves, extending to the chordæ tendineæ, which are frequently eroded and ruptured, and sometimes penetrating deeply into the myocardium.

The vegetations are usually very irregular in arrangement, contrasting greatly with the more or less limited distribution of those in simple endocarditis. They frequently extend to the mitral endocardium, and particularly is this the case when the chordæ tendineæ of the anterior flap of the mitral are ulcerated through, abundant vegetation being then usually present on the posterior and outer part of the wall of the left auricle. Occasionally the vegetations extend upwards along the aorta and pulmonary artery, and sometimes encroach upon the orifices, and even spread into the channels of the coronary vessels.

Mural malignant endocarditis without involvement of the valves is almost unknown. Occasionally an aneurysm of a valve is produced. Sometimes foci of suppuration occur in the valves, or small abscesses may form in the subendocardial and myocardial tissues. In this way cardiac aneurysm or even penetration and rupture of the heart may result.

Secondary and Associated Lesions.—But the lesions noted in cases of malignant endocarditis must be carefully distinguished accordingly as they (i.) are primary and have preceded the endocarditis, or are (ii.) secondary and due to or associated with it. In the latter cases they will usually be the result of (1) embolism or (2) toxic absorption.

The secondary lesions include infarction of the lungs, spleen, and kidneys; cerebral embolism and softening; embolic blocking of mesenteric vessels or peripheral arteries; and multiple hæmorrhages into the skin, serous membranes, and retina.

Congestive and degenerative states of various organs are common. The spleen is usually much enlarged. The kidneys also frequently present profound tubular changes. Infarcts manifest much caprice in their distribution. It is, however, exceptional for the secondary embolic centres to become the seat of suppuration.

Reference to some of the conditions noted in the 65 fatal Manchester cases may not be without interest. Infarction occurred in 34. The spleen was involved in 20, the kidneys in 11, the lungs in 7. In one instance an embolus blocked the right middle cerebral, in another the femoral, and once an embolic aneurysm of the coronary artery was found. Acute lobar pneumonia existed in 12. In the majority of these it was apparently secondary, and possibly in several of embolic origin. Hæmorrhage into the lungs existed in a number. Acute pleurisy was marked in 7, and in 3 empyema had occurred. Acute pulmonary tuberculosis, with extensive excavation and tuberculous ulceration of the intestines, was met with once. Acute pericarditis was found in 4, and old adherent pericardium also in 4. Peritonitis was present in 3, one of these being evidently tuberculous. Gastric ulcer of recent formation occurred once. In two instances a duodenal ulcer had lead to death from hæmorrhage. In another case intestinal ulceration existed, possibly of embolic origin. Suppurative cholecystitis was found twice; in one the gall-bladder was obstructed by old inflammatory thickening, but no calculi were present. Hepatic abscesses were noted once, and in the same case there was extensive abdominal tuberculosis. Well-defined hepatic cirrhosis was present in at least 4. Most cases had a more or less "nutmeg" condition of the liver. Splenic enlargement was a special feature in 53. In another case which had appeared during life to be one of "splenic anæmia," though there had been prolonged inter-

mittent pyrexia, the spleen was of enormous size. The average weight in 55 cases was a little over 16 oz. The great variation in size and weight will be seen from the following figures:—One weighed 70 oz., 2 over 30 oz., 15 between 20 oz. and 30 oz., 24 between 10 oz. and 20 oz. In a few instances the spleen was not appreciably enlarged post-mortem. Nephritis mainly of a parenchymatous type existed in 11 cases. "Granular" kidney was met with once. Acute septic meningitis was noted twice. In another case puriform fluid was found within the lateral ventricles. Cerebral softening existed in 3, in 2 being manifestly of embolic origin. Meningeal hæmorrhages occurred twice. Extensive cerebral hæmorrhages were present in 3, and in only one could a distinct embolus be found. Embolism of the femoral occurred once. Pus in the joints was present twice, and boils were also noted in 2 cases.

Morbid Histology.—The action of the micro-organisms leads to changes which are partly (i.) necrotic, but in other portions (ii.) inflammatory.

Where the endocardium is far removed from vessels, as in the greater part of the valves, unless sclerosed, the former process is predominant, but where the microbic invasion occurs in proximity to vascular regions an inflammatory exudation speedily follows.

The action of the organisms or their toxins leads at first to rapid degeneration and later to molecular death of the infected tissues, and the chemico-physical changes in the affected part produce clotting over the diseased structures. In vascular regions extensive inflammatory exudation occurs. The leucocytes are collected sometimes in sufficient numbers to constitute distinct areas of suppuration. In less acute cases proliferation of the connective-tissue elements occurs. The endothelium and portions of the necrosed tissue are readily detached, and the projecting cellular elements becoming the seat of deposits of fibrin form irregular, soft vegetations. By the separation of the dead tissue and the detachment of the vegetation ulcers are produced. Ulceration and the formation of vegetations are usually met with in association.

Suitable staining reagents demonstrate the presence of the infecting micro-organisms in the affected parts.

Post-mortem examination of these cases should include a full and complete bacteriological investigation of the blood, vegetations, splenic pulp, and secondary foci when present. Every care must be taken to prevent any extraneous contamination. Particulars as to technique will be found in the ordinary works on bacteriology.

Pathological Chemistry.—Recent researches have shown that the products of the growth of certain organisms taken from cases of infective endocarditis are of the nature, the one of a proteid consisting of proto-albumose and deutero-albumose, and the other, a non-proteid body with strong acid reaction.

II. Valvular Affections

Before considering the various morbid conditions producing inefficiency of the individual valves, reference may be made to certain general pathological features; for it is only by the painstaking comparison of clinical phenomena with the results of pathological investigation, that mere vague generalisations or inaccurate deductions can be successfully overturned.

Etiology.—The precise nature of the influences predisposing to or exciting valvular disease is in only too many instances far from clear. In some the morbific influence operates during intra-uterine development.

The agencies producing valvular disease are frequently so combined as to render a strict differentiation into predisposing and exciting influences impossible. It would seem that in many instances a distinctly extraneous agent can only exert its pathogenic power when assisted by intrinsic conditions tending to lower the general or local resistance of the body, or to increase the physiological work of the part affected. Undoubtedly infective agents hold an important position as excitants, and there is every reason to believe that toxic bodies, either of autogenetic or heterogenetic origin, play an important part in the etiology of valvular disease of the heart.

Heredity.—Family transmission of a tendency to valvular disease may be admitted as a possibility, but acting probably by the continuance of constitutional disease or hereditary depravity of tissue. The importance of the vascular system, and with it, of necessity, the central organ in relation to heredity, cannot be doubted. The family connection of coronary disease and angina pectoris is clearly established. States of high arterial tension also seem to "run in families," and several children of the same parents may suffer from an apparently similar form of cardiac inadequacy. Race, as far as is known, is not of particular etiological importance.

Sex plays a determining part, as indicated by the fact that mitral stenosis is commonest in women, while aortic disease is most frequent in men. This influence of sex would seem to be mainly dependent on exposure to exciting conditions; thus rheumatic affections are undoubtedly most usually met with in females; while males, by their occupation or exposure to agencies producing arterial and muscular degenerative changes, are most liable to valvular defects from an aortitis implicating the function of the valves in one way or another.

Age.—Valvular defects, as already indicated, may originate during intra-uterine life. During the period of normal development, childhood, and early adult life, morbid processes of an inflammatory nature, and usually rheumatic in

origin, are particularly prone to affect the valves. Microbic valvulitis, or an endocarditis dependent on invasion by infective organisms, may occur at any age, but is most frequent in adults. Auriculo-ventricular valvular defects arising from primary muscle failure are usually met with in mature subjects, and generally at what should be the period of maximum vigour. In advanced and declining years the aortic valves are peculiarly prone to suffer from degenerative changes, and are liable to be involved by low inflammatory and degradation processes extending to them from adjacent parts. Thickening of the auriculo-ventricular segments is not infrequently met with by the pathologist when there had been no clinical evidence of impairment of their action. In females the period of sexual activity, with its elevations of arterial tension and the strain of child-bearing, is of considerable importance in advancing a valvular condition practically latent to one associated with marked symptoms.

Temperament is a factor which may be considered of some slight etiological importance in accelerating or retarding the day of cardiac failure. The high tension engendered by worry is apt to be neglected.

Previous Disease.—As already indicated, a valve in any way impaired, either by congenital deficiencies or acquired defects, is prone to become the seat of further morbid processes, and this would seem to be particularly the case when the exciting agent is of an infectious nature.

Climate.—The manifold factors included under this term can only be considered of importance in so far as they influence the production of diseases, like rheumatism, which are intimately allied with morbid processes occurring in the valves. The relation of Bright's disease to climate must also be noted, for the connection of renal affections and certain forms of cardiac disease is a close one.

Social position becomes of importance in influencing exposure to definite morbific influences. The inability of the poor to obtain a livelihood without hard work means early failure to many a cardiac cripple.

Occupation must be admitted as not only a predisposing factor, but oftentimes a contributing influence. The importance of work necessitating stress and strain cannot be set aside. Its action is complex: sometimes it may act through sudden injury, but more frequently it leads to valvular deficiencies by the establishment of chronic inflammatory processes, causing aortic dilatation or direct implication of the valves, while there may be at the same time muscle failure brought about by coronary interference or otherwise.

Education, or rather its associated methods, unfortunately cannot at present be excluded from the group of predisposing causes. There is reason to believe that mal-education, at least in physical procedures, oftentimes precipitates a cardiac breakdown in young subjects, who, perhaps, with slight valvular impairment following rheumatic endocarditis, are quite unfitted for specially trying forms of drill, exercise, or athletic pursuits. Still, taken altogether, the influence of athletics is beneficial; the good outweighs the evil.

Habits are of importance, as, for example, alcoholism, sexual excesses, depraved feeding, etc.; but reference to these will be found in the clinical section.

Exciting Causes.—Among the specific influences productive of valvular disease *rheumatism* stands out pre-eminent. As to its precise nature we are still in the dark. We may safely assert, however, that by the prevention of rheumatic fever, and such "rheumatic" manifestations as chorea, rheumatic arthritis, and "growing pains," the greater number of chronic valvular affections would be effectually banished. In every case of chronic valvular disease a history of rheumatism, or the so-called "rheumatic" affections, should be sought. Several of the infectious diseases are said occasionally to give rise to progressive valvular endocarditis. Possibly scarlet fever may sometimes so act; but even here the relation to rheumatism must not be lost sight of, for there are several quite distinct forms of arthritis met with in scarlatina. Septic and other organisms, as previously indicated, may establish or continue valvular disease, and usually such proceed more or less rapidly to a fatal issue; but some pathologists contend that death of the microbes and subsequent cicatrisation of the valves occasionally occur. Such a view is exceedingly difficult, if not impossible, to prove.

Both autotoxic and heterotoxic agents seem capable of initiating chronic inflammatory and degenerative changes in the fibrous and muscular elements of the heart, and in the effects of which the valves may possibly participate. Syphilis, gout, and Bright's disease must here be mentioned. Syphilis, however, although acting disastrously on the coats of the aorta, so leading to dilatation and valvular incompetence, does not commonly affect the valves themselves. One form of localised supra-valvular aortitis, of which we have met with several examples, is probably syphilitic in origin, and has a special tendency to produce obstruction of the coronary arteries and extension to the aortic valves, rendering them incompetent. Gout, alcohol, and Bright's disease probably never give rise to valvular disease directly. Gout, and with it plumbism and Bright's disease, are of course associated with high arterial tension and implication of the vascular system, and thence secondary valve implication or incompetence may arise.

The etiological importance of mechanical strain has already been insisted upon, and direct

or indirect violence must also be admitted as a possible, although, under normal circumstances, most unlikely cause of valvular deficiency.

Morbid Anatomy.—A clear comprehension of the gross lesions affecting the valves of the heart will do much to simplify the clinical manifestations, and to elucidate the consequences and associated changes of valvular disease.

As already indicated, the valves of the left chamber of the heart are most commonly affected. It is usually only in fœtal and infective endocarditis that the right side is alone involved. In simple as well as in malignant endocarditis both sides of the heart may suffer, but even then the involvement of the left is usually most extensive. Double auriculo-ventricular stenosis is not very rare, but when met with the left-sided lesion is always far in advance of that of the right.

The valves may suffer primarily, or become impaired only secondarily. The exact mechanical deficiency predominant may be more or less of an accident pathologically speaking, and between the huge orifice of a highly incompetent mitral orifice and the chink-like lumen of an extreme stenosis almost every gradation may occur.

Stenosis of an orifice is produced by the thickening and contraction of the valve segments, by their adherence to each other, to the thickening, shortening, and fusion of adjacent chordæ tendineæ, by deposition of lime salts in the structure of the valve increasing its rigidity, preventing close apposition of segments, and forming a narrow yet possibly ever open door.

Occasionally aneurysmal dilatation of the valves also produces some degree of obstruction, but this is not so frequently the case as in acute endocarditis, and the condition is rather a pathological curiosity than a fact of practical importance.

Incompetence may arise from defects in the valves themselves such as above indicated, or may be secondary to deficient systolic narrowing of the orifice because of muscle failure; or inefficiency of contraction of the musculi papillares may interfere with the control normally given to the valves by the chordæ tendineæ. Indeed, in considering mitral lesions it is impossible to eliminate the effects of associated muscle failure from the causes at work in the production of the valvular incompetence.

Morbid Histology.—The essential features of chronic disease of the valves consist in the production of a fibrous cicatricial-like tissue, in which calcareous salts are peculiarly prone to be deposited.

Consequences and Associated Changes.—The effects of valvular disease may be divided into (i.) intra-cardiac and (ii.) extra-cardiac. It is only necessary to mention them here as they are referred to elsewhere.

1. Alteration in the size, weight, shape, and relative proportions of the different parts of the heart.
2. Extension of sclerosis to the adjacent endocardium and formation of localised thickenings and irregularities.
3. In some instances diminished blood-supply, with consequent anæmia and impaired nutrition.
4. Disturbances of the pulmonary circulation.
5. Distension of the venæ cavæ and their tributaries.
6. Intra-cardiac clotting with formation of cardiac thrombi.
7. Formation of emboli.
8. Hæmorrhage from simple engorgement of vessels apart from embolism.
9. Dropsy.
10. Blood-changes.

Affections of the Aortic Valves and Orifice

Aortic disease dependent on valvular lesion may occur:

1. By primary involvement.
   - (i.) Of acute development.
     - (*a*) External violence.
     - (*b*) Rupture.
     - (*c*) Malignant endocarditis.
   - (ii.) Of slow development.
     - (*a*) Maldevelopment.
     - (*b*) Chronic valvular endocarditis.
     - (*c*) Degradation processes.
2. By secondary involvement.
   - (i.) Of acute development.
     - (*a*) Extension of acute aortitis.
     - (*b*) Extension of malignant endocarditis.
   - (ii.) Of slow development.
     - (*a*) Extension of chronic aortitis.
     - (*b*) Extension of chronic endocarditis.

Etiology.—Disease of the aortic region of the heart impairing the efficiency of the valves is essentially an affection of adult men, although, of course, it is not infrequently met with in young subjects of both sexes in the form of an infective endocarditis, or as a consequence of a rheumatic affection.

It is the aortic valves which are most markedly exposed to and deteriorated by the stress and strain of excessive or unregulated physical exertion. Under such circumstances disease of the aorta has generally preceded the crippling of the valve segments, and having accomplished their incompetence by causing dilatation of the vessel by virtue of impairing its elasticity, and throwing strain upon it during the ventricular systole. A rheumatic affection of the aortic valves is usually met with in association with involvement of the mitral segments, but in a certain number of instances the aortic valves are the only ones seriously impaired. In the production of the crippling sclerosis which accounts

for such a large number of cases of aortic deficiency in addition to strain, gout and Bright's disease are often said to be of etiological importance, but they are so probably only through the influence of vascular pathological states. Certainly sclerotic valves are commonly met with in subjects who have never experienced the toxic influences of gout nor suffered from Bright's disease, while on the other hand chronic endocarditis probably never occurs as a direct consequence of these states. Malaria has been credited with being instrumental in establishing a chronic aortitis. All conditions inducing high arterial tension must be considered as agencies making for strain of the aortic valves. Such occupations as those of miner, collier, smith, soldier, and the like, undoubtedly predispose to aortic disease. Syphilis, however, must also be admitted as a factor in some of these cases.

External violence and strain may, under exceptional circumstances, lead to sudden rupture of an aortic valve. In some of the recorded cases there is reason to believe tearing occurred in a valve already weakened by existing disease. The valvular laceration occurs during severe muscular exertion, and is immediately followed by more or less cardiac pain and distress, and usually marked dyspnœa.

Extensive mechanical defects often result from malignant endocarditis. The abundant flocculent vegetations may tend to occlude the orifice, or the formation of a valvular aneurysm may produce a degree of obstruction, but more frequently extensive ulceration leads to a rapidly established incompetence permitting of free regurgitation.

Maldevelopment or disease during intra-uterine life is a rare cause of aortic disease.

It is well to note that it is by no means rare to find a thin fringe of fine bead-like vegetations in a rheumatic case along the ventricular aspect of the cusps, which during life had presented no clinical evidences of mechanical deficiency.

Chronic aortitis is one of the most prolific agencies by which aortic valvular disease is produced. From the clinical point of view the actual form of aortitis is of comparatively little importance. Most frequently it is of an atheromatous variety. The aortitis deformans may lead to incompetence without directly involving the valve segments by producing dilatation of the aorta, and ultimately an increase in the size of the aortic ring, but often there is also stiffening and thickening of the cusps.

Reference may here be made to a special form of aortic disease leading to a certain degree of incompetence, and usually producing considerable impairment of the cardiac nutrition by narrowing the orifices of the coronary arteries. The condition involves most markedly the aorta immediately above the valves. It consists of a chronic localised aortitis, and occurs usually in patches, the borders having often an undulating or serpiginous outline very suggestive of a syphilitic process. In the cases we have examined, however, no very distinct syphilitic lesions could be detected elsewhere in the body. In the case of a young woman where this condition existed, and where angina pectoris was a prominent symptom, pelvic inflammation had existed, and there were other reasons to believe syphilitic infection had occurred.

Morbid Anatomy.—Brief reference may be made to the lesions found :—

1. *In Aortic Incompetence*.—In cases of aortic regurgitation due to chronic endocarditis and sclerosis of the valve segments, conditions are found which in most instances are the result of an association of processes. There is also oftentimes considerable variation in the seat and extent as well as in the predominant character of the morbid conditions.

In some instances the portion of the cusp nearest the base of attachment is indurated, and it may be contracted while the remaining portion is practically uninvolved. Here the normal part may be retroverted or doubled backwards towards the ventricular cavity in consequence of the failure of the segments by approximation to sustain the aortic blood-pressure during diastole.

Sometimes the segments are merely uniformly indurated. They may be irregularly thickened, puckered, and extensively deformed. Oftentimes the distortion more particularly affects the free edges of the flaps, which may be rounded and everted or rolled towards the base of the aorta. Occasionally one of the cusps appears as though dragged away from its attachment, and droops ventricle-wards. Adhesions are sometimes formed between adjacent flaps, and when these are broken through a large pouch may be formed by the combined cusps. We have met with distinct aneurysmal pouching in a sclerosed valve.

In many of the cases where calcification of the valves is a conspicuous feature the aorta is practically healthy. This would seem to indicate that the valves had been affected primarily. When of rheumatic origin there is a particular disposition to the deposition of lime salts. In the past there has been too great a tendency to consider calcareous infiltration as evidence of the process loosely termed "atheroma."

Where calcification is a conspicuous feature, it is well to observe that the deposit of lime salts is often not limited to the valves, but may be abundant in the infra-valvular portion of the septum and in the anterior segment of the mitral.

Incompetence may arise from dilatation of the aortic ring without any distinct involvement of the valve segments themselves.

2. *In Aortic Stenosis*.—Chronic rheumatic endocarditis is particularly influential in leading to aortic stenosis.

Pure cases of aortic stenosis are by no means common, although a certain degree of obstruction

exists in a large number of the cases in which incompetence is the conspicuous lesion.

Aortic stenosis is always due to valvular disease. The actual lesion presents considerable variation in its exact form, seat, and extent.

Sometimes the whole of the cusps are so infiltrated with lime salts as to be separately indistinguishable, and form a calcified ring, in the centre of which is a mere chink of a lumen. Or the valves may be thickened, fused one to the other so as to constitute a kind of funnel with its narrow end or apex projecting into the aorta.

Frequently the segments are irregularly involved. In some cases where there is abundant deposition of lime salts, intervening portions of fairly healthy valve tissue may sometimes be observed. Commonly the valves are thickened and rigid, coherent, and, according to some pathologists, rendered rough by the calcification of adherent thrombi, the lumen being often so narrow as to barely admit a goose-quill.

3. *Aortic Incompetence with Stenosis.*—From what has already been said it will be clear from the pathological necessities of the case that mixed or combined conditions must occur. Such is commonly the case. In a considerable number of instances where incompetence is the conspicuous clinical feature some degree of aortic obstruction will be found post-mortem.

Affections of the Mitral Valves

The nature of the processes leading to deficiency of the mitral valves does not differ from those already described as occurring in valves generally, or in the aortic segments in particular. It will, therefore, only be necessary to refer to points of special practical importance. The following forms of mitral disease may be distinguished:—

1. Mitral stenosis.
2. Mitral stenosis with incompetence.
3. Mitral incompetence.

Although doubtless in most cases in some stage of a chronic primary valvular disease of the mitral segments combined mechanical defects occur, yet for purposes of practical discrimination, as well as for convenience of grouping and description, the above three classes may be recognised. It may, nevertheless, be best to add at once that in nearly all cases of mitral stenosis incompetence of a greater or less degree takes place sooner or later.

Mitral Stenosis.—Left auriculo-ventricular obstruction forms one of the most important of the organic diseases of the heart. It occurs in the majority of cases associated with some incompetence.

Etiology.—Mitral stenosis is particularly common in females, although met with also in a considerable number of males. While often not coming under medical observation till adult life, and sometimes not until the latter part of middle life, the condition is generally initiated at an early age; the largest number of cases are met with before the age of thirty. The great majority of cases are due to a chronic endocarditis. Sometimes this would seem to arise as an acute condition. Rheumatic fever or chorea are the common antecedents. In some instances no definite excitant can be detected. Where no distinct rheumatic history is forthcoming careful inquiry will frequently elicit some reference to "growing pains" or chorea.

In some very rare instances mitral obstruction has been dependent on the abundant formation of vegetations, or the intrusion of a so-called cardiac polypus or tumour.

Morbid Anatomy.—Mitral stenosis is the result of very conspicuous organic changes in the valves. The auriculo-ventricular ring and the structures in immediate connection may be involved. Occasionally the narrowing of the mitral orifice appears to be mainly, and according to some even entirely due to an induration with contraction, and sometimes calcareous deposition in the fibrous ring encircling the auriculo-ventricular orifice and the base of the valve, as well as in the immediately adjacent muscular tissue. Most usually the stenosis is due to extensive changes in the valve segments. The sclerosis and cicatricial formation is apparently the result of a chronic inflammatory process. The valve segments, and usually the anterior or aortic cusp is the one principally involved, are much thickened, of firm and oftentimes cartilaginous consistency, and frequently puckered, shrunken, or otherwise deformed. A variable degree of calcareous infiltration is of common occurrence. The chordæ tendineæ are usually greatly thickened, very rigid, shortened, and often fused or firmly adherent in their course to the valves, forming stiff ridges of dense fibrous tissue, or actually incorporated into the sclerosed funnel. The apices of the musculi papillares are frequently converted into dense white cartilaginous-like tissue. The lumen of the auriculo-ventricular orifice is of course narrowed. A normal adult mitral orifice should readily admit two finger-tips. A stenosed mitral orifice frequently will only allow of the insertion of the tip of the forefinger, and sometimes the degree of stenosis is still greater.

According to the structural deformity two forms of mitral stenosis have been described: (i.) the button-hole form, and (ii.) the funnel-shaped variety. In the former the mitral orifice presents the appearance when viewed from its auricular aspect of a slit in a tough and rigid diaphragm. In the latter the contracted cusps, with fused chordæ tendineæ projecting ventriclewards, give the aspect of that of a funnel. Often, however, what looks like a button-hole variety when viewed from above will assume a more or less typical funnel-shape appearance when viewed from below. There would, there-

fore, seem to be no clear pathological reason or clinical advantage in insisting on this somewhat artificial division.

It is well to remember that in many cases of mitral stenosis the natural seat of the auriculo-ventricular orifice is shifted, and instead of being on a level with the base of the valve segments, is lowered to a position at the apex of the funnel-shaped rigid valve.

Still further, the obstruction is occasionally increased by the formation of thrombi on the aricular surface of the sclerosed valve, or by pendulous coagula from the left auricle.

The results of this lesion on the general circulation have already been sufficiently described, and certain of the local effects will be referred to presently when considering hypertrophy and dilatation, so that it is only necessary here to mention the more conspicuous intra-cardiac consequences and associations. Naturally the chief stress of mitral stenosis falls on the left auricle, which, according to the stage of the lesion, presents variable degrees of hypertrophy and dilatation.

There is also considerable increase in the size of the right ventricle, the hypertrophy of its walls and prominence of its infundibulum being usually most conspicuous.

In the early stages hypertrophy is the chief if not sole feature, but in old-standing cases, where a certain amount of mitral incompetence has existed with progressive heart failure, more or less dilatation is usually found at the autopsy. Possibly some degree of incompetence has much to do with this. The auricular appendix is often blocked by firm thrombi, and the cavity of the auricle generally contains much A.M. and P.M. blood-clot.

Theoretically it might be considered that the left ventricle should show but little alteration, or even be diminished in size, but generally when examined post-mortem it presents some degree of dilatation, and not infrequently distinct hypertrophy.

Mitral Incompetence.—In the normal heart the mitral valve effectually prevents any communication between the left ventricle and the left auricle during the systole of the former.

Two distinct forms of mitral incompetence, essentially different in etiology and mechanism, although presenting many clinical features in common, can be recognised:—

(i.) Incompetence from primary valvular disease.
(ii.) Incompetence from muscular failure.

Etiology.—According to the form of mitral incompetence, two chief groups of etiological agents are to be sought:—

1. That in which the curtains of the mitral valve or their tendinous cords are affected.

(i.) Infective Endocarditis.—The rapidly developed and often very extensive and destructive character of this malignant inflammatory process has already been indicated. The mitral segments, like the aortic, frequently suffer, and very extensive incompetence may be established, and that so speedily as often to give little opportunity for any effective compensatory changes.

(ii.) Simple acute endocarditis, generally of rheumatic order, although often considered as giving rise to regurgitation, probably often allows of such rather from the associated muscle failure than the actual changes occurring in the valve segments—at least the muscle failure assists in the result.

(iii.) Chronic endocarditis, the consequence usually of rheumatism as already explained, not only leads to deformities, but is apt to produce loss of mobility, and a deterioration of the structures as an efficient mechanical valve. Although sclerosis of the mitral tends to produce incompetence, and that perhaps at a comparatively early stage in the cardiac history, yet in many instances the degree of compensation has been such that the case does not come under medical observation until the mitral orifice has been considerably stenosed, and the then marked regurgitation is obviously occurring through an obstructed orifice. In all probability, however, some degree of regurgitation invariably occurs before the development of stenosis.

(iv.) Traumatic influences are said to be capable of establishing sufficient injury to the mitral valve as to produce incompetence, but it is difficult to obtain evidence in favour of such an assertion.

2. That in which the valve curtains are normal, but changes in the muscle substance lead to a widening of the mitral orifice. In advanced cases probably also some yielding of the structures forming the auriculo-ventricular ring may occur.

Mitral regurgitation from such causes may be of almost any degree of severity. In some the enlargement of the mitral orifice is slight and temporary, in others it is enormous and persistent. One group may be relatively curable; another incurable and incapable of lasting amelioration.

All conditions leading to a lowering of the nutrition of the cardiac muscle are to be considered instrumental in the production of this form of mitral insufficiency. Among such are the following: (i.) Febrile states; (ii.) Toxic conditions; (iii.) Anæmic affections; (iv.) Local inflammatory degenerative or other abnormal processes affecting the myocardium; (v.) Some chronic wasting diseases.

It may be well here to point out more explicitly that the toxæmic influence of many of the infectious diseases on the cardiac muscle may be such as to effect marked mitral insufficiency. Rheumatism, also, although attacking the endocardium in chief degree, can sometimes produce such myocardial enfeeblement as will lead to a considerable amount of dilatation, and amply to account for signs of mitral incom-

petence. The importance of muscular strain in such states as those above indicated, or indeed in any condition of cardiac debility, is self-evident.

Mitral incompetence due to gross lesion of the valve itself is, of course, strictly speaking, incurable, although adequate cardiac action, if compensation be established, may be maintained for years. Incompetence from muscle failure, on the other hand, may be to all intents and purposes curable, provided that the influences productive of the muscle failure can be removed and the state of normal nutrition re-established.

Morbid Anatomy.—As already indicated, two distinct classes are to be recognised:—(i.) Incompetence from primary valvular defect; (ii.) Incompetence from muscular weakness.

The valvular defects are such as have been previously described. They consist of a thickening, puckering, and shrinking of the curtains of the mitral, with usually more or less shortening, thickening, and induration of the tendinous cords. In a very considerable number of the cases which *clinically* presented the classic indications of mitral incompetence narrowing of the mitral orifice is found.

The muscle failure may manifest itself pathologically by dilatation of the ventricular cavity and simple enlargement of the auriculo-ventricular orifice, but these conditions will be best referred to later.

Affections of the Pulmonary Valves

Lesions limited to the pulmonary valves and orifice are rare. Sometimes, however, the pulmonary artery and valves are involved in association with or secondary to affections of the left side of the heart.

Two conditions are of clinical importance:—

(i.) Pulmonary stenosis; (ii.) Pulmonary incompetence.

Pulmonary Stenosis. — Obstruction at the pulmonary orifice may be of (i.) congenital origin, or (ii.) acquired subsequent to birth.

Congenital pulmonary stenosis constitutes one of the commonest varieties of cardiac malformation. It is frequently associated with imperfection of the auricular and ventricular septa and patency of the ductus Botalli. All degrees may be met with up to complete occlusion of the orifice and atresia of the pulmonary artery.

It is often difficult, both clinically and pathologically, to distinguish between a stenosis of congenital or acquired origin.

Primary pulmonary stenosis, arising subsequent to birth, is exceedingly rare. When met with it is usually dependent on old endocarditis of rheumatic nature. In not a few of the recorded cases the etiology has been obscure.

The valves are thickened, often rigid, and even infiltrated with lime salts. The edges of the cusps may be fused and a funnel-shaped channel so produced, the apex directed upwards towards the pulmonary artery. The orifice may be diminished to a mere slit. Vegetations may be present, and so still further narrow the orifice.

Occasionally in malignant endocarditis large fungating vegetations form on the pulmonary cusps and partially obstruct the orifice.

Obstruction has been met with at a point beneath the pulmonary valves. The walls of the conus arteriosus are thickened, rigid, and contracted, as from old ventricular endocarditis. There may be recent vegetations about the indurated region.

Obstruction has also been known to arise from chronic atheroma of the pulmonary artery.

In all cases of pulmonary stenosis the right ventricle will be found hypertrophied, and usually with its cavity dilated.

The pulmonary artery, as already indicated, while generally involved in the congenital cases of stenosis, does not usually present much change in the acquired form.

Pulmonary Incompetence.—Incompetence of the pulmonary valves may arise from the following causes:—

(i.) Primary disease of the valve segments. This may occur: (*a*) in pulmonary stenosis; (*b*) chronic endocarditis and sclerosed conditions of the sigmoid cusps unassociated with stenosis; (*c*) in malignant endocarditis; and (*d*) in the very doubtful cases of laceration from trauma.

(ii.) Secondary involvement of the valve: in (*a*) dilatation of the pulmonary artery; (*b*) pressure on the orifice and valves from without as very exceptionally in aortic aneurysm.

It will thus be seen that the mechanical defect of pulmonary leakage may arise from several causes. In some instances the condition is undoubtedly of congenital origin, but it is then usually associated with obstruction of the orifice.

Probably the so-called simple pulmonary endocarditis is of rheumatic origin. In not a few cases, however, of advanced aortic and mitral disease we have found a certain degree of induration of the pulmonary segments, and it seems quite feasible to believe that this thickening is due to excessive pressure, and is allied to atheromatous disease of the aorta.

Simple valvular disease of the pulmonary cusps presents the usual appearances of a chronic, inflammatory, and degenerative process. The segments are indurated, puckered, and do not approximate, but allow of free leakage.

The right ventricle and right auricle become hypertrophied and dilated. The pulmonary artery may be normal in size, dilated, or even contracted in some cases.

A relative or functional incompetency of the pulmonary valves may occur as a natural adaptation, as in cases of persistent high pulmonary pressure.

In advanced mitral stenosis, however, the

pulmonary artery is more or less constantly distended, and dilatation occurring, may allow of regurgitation through the pulmonary orifice. In such cases, also, the pulmonary artery may be atheromatous.

Affections of the Tricuspid Valves

Primary or isolated lesions of the right auriculo-ventricular valves are exceedingly rare, while secondary impairment of their mechanical action is met with in the greater number of cardiac cases.

During fœtal development malformations and inflammatory processes are more liable to involve the tricuspid than the mitral valves, but after birth, although the tricuspid may suffer in connection with or secondary to the other valves, a lesion limited to them is quite exceptional.

The morbid conditions may be divided into (i.) Tricuspid stenosis and (ii.) Tricuspid incompetence. When the former exists the latter is also usually present.

Tricuspid Stenosis.—Tricuspid obstruction ranks amongst the rare forms of valvular lesion. The greater number of recorded cases have occurred in females. The age at death is usually between 20 and 30. It is often said to be of congenital origin, but of this we have grave doubts. In most instances it is associated with mitral stenosis. As a general rule the degree of stenosis of the tricuspid is much less than that of the mitral. In most of these cases the involvement would seem to be coincident and dependent on rheumatism. Tricuspid stenosis is also said to arise as a secondary lesion from inflammatory processes set up by back-pressure acting through the pulmonary circuit. It is probable that this is never the case.

The morbid anatomy of the stenosed tricuspid is almost identical with that of the similarly affected mitral, and hence does not call for any detailed description. The cusps are thickened, contracted, more or less adherent to one another, deformed, and sometimes connected into a funnel-shaped structure with induration and adhesion of the chordæ tendineæ, and the orifice thereby greatly narrowed. Vegetations may still further obstruct the orifice, and abundant deposition of lime salts may add to the rigidity of the sclerosed tissues.

The secondary results have already been sufficiently indicated, and need not be further dwelt on, except to point out that the greater part of the changes found in the chambers of the right heart are, generally speaking, due rather to the usually present and more important mitral stenosis than the tricuspid lesion.

Tricuspid Incompetence. — Insufficiency of the tricuspid valves, allowing of regurgitation, is, as above indicated, a common valvular defect. Indeed, in states of over-distension, such as may occur in the course of violent exertion, a leakage or "safety-valve" action may be considered as physiological.

For practical purposes, therefore, it is all-important to differentiate cases into two groups:—

(i.) Tricuspid regurgitation arising from muscle failure of the heart and independent of lesion in the structure of the valves.

(ii.) Tricuspid regurgitation dependent on morbid conditions of the valve segments.

Where the latter exists, the influence indicated in the former is only too apt to be sooner or later superadded.

Tricuspid regurgitation from relaxation or stretching of the fibrous ring constituting the base of the valve undoubtedly occurs, but usually has been for long preceded by asthenic or distinctly morbid affections of the muscular substance. Conditions of degeneration, inflammation, or like states impairing the contractile power of the cardiac walls and papillary muscles, arise from a variety of agencies, as already indicated, and hence it is easy to understand how tricuspid incompetence may be consecutive to many different diseases and dissimilar lesions.

It is necessary also to insist on the paramount importance of influences acting on the muscle of the right ventricle through the pulmonary system. This arises either from local morbid conditions of the lungs, such as chronic bronchitis, emphysema, fibrosis, and the like, or from backworking of the effects of left-sided lesions and conditions of high arterial tension such as occurs in chronic Bright's disease.

But in a considerable number of the cases of tricuspid incompetence there is a distinct alteration in the valve segments. In one class the cusps will be found thickened, puckered, shrunken, and with induration of the chordæ tendineæ, and possibly calcareous deposition. Usually the mitral will also be involved to a greater extent, and frequently the aortic cusps. Here the valvular crippling is dependent, in the great majority of cases that are not congenital, on rheumatism or chorea.

Atheroma is sometimes said to involve the tricuspid valves, but we believe this to be quite exceptional.

In another class of cases the tricuspid valves become swollen, deformed, and more or less destroyed by a malignant endocarditis.

Tricuspid regurgitation being generally a secondary condition, the state of the heart will often be in great part dependent on the primary lesion, but in the cases in which the tricuspid incompetence is of primary origin, or at all events the most conspicuous feature, the right auricle will be found enormously dilated with a variable degree of hypertrophy of its walls, although sometimes they may be distinctly thinned. The venæ cavæ are usually much

distended. The general effects have already been fully indicated.

III. Affections of the Myocardium

It is only of recent years that a true appreciation of the importance of lesions of the myocardium has aroused pathologists and clinicians to a more intimate study of the same. A clear recognition of anatomical arrangements and structural peculiarities constitutes an essential preliminary. Of special importance is the vascular supply. The structure and characters of the coronary vessels have, however, been described elsewhere and need not be referred to again, except to insist on their great importance in considering many of the most important pathological conditions of the myocardium.

In dealing with the processes of disease occurring in the heart frequent reference has been made to involvement of the muscle substance. It will be well, therefore, in the present section to limit our consideration to the more important affections of the myocardium.

Cardiac debility, although a term which, pathologically speaking, may be considered indefensible, nevertheless conveniently indicates a condition, congenital or acquired, of inadequacy without visible structural change. Asthenic states of the cardiac muscles arise often under the influence of deficient coronary arteries, and may even end in angina pectoris. Sometimes cardiac debility is temporary, but more frequently it is of long duration, and constitutes one of the forms of disability embraced under the comprehensive but indefinite clinical designation of "weak heart."

Atrophy and degenerations of the myocardium are very common. They are to be looked upon as evidences of either local or general malnutrition. In not a few instances the muscular degradation is dependent upon general blood states of anæmia or toxæmia. In other local conditions involvement of the coronary vessels is the determining cause.

Simple atrophy is said to occur as a part of the general wasting incident to old age, but certainly in healthy old people the very opposite seems frequently to be the case. Atrophy arises also in conditions of starvation and general malnutrition, and as a part of a more direct disease, such as fibrous transformation of the myocardium.

Special forms of atrophy have been described, such as:—

(i.) Fibrillary atrophy, where there is, as it were, an exaggeration of the longitudinal striation of the muscle fibres;

(ii.) Fragmentary atrophy or segmentation, in which a splitting up of the muscle cells is noticed, probably from a necrosis of the myosin; and,

(iii.) Pigmentary atrophy, where hæmatoidin granules of a brownish-yellow colour appear about the nuclei of the muscle cells, and the myocardium becomes darker, harder, and lighter in weight.

Degenerations of varying degree are common, but have been already sufficiently described.

Myocarditis or inflammation of the cardiac muscle is practically always a secondary condition. It may develop in the course of infectious fevers or in association with septic and toxic states. It is also said to arise from a local extension, as from endocarditis or pericarditis.

Myocarditis is often described as being either (i.) parenchymatous or (ii.) interstitial; but such a distinction is somewhat artificial, as in most instances both muscle and inter-muscular substance are involved. The process may be (i.) general or (ii.) local. As regards intensity it is (i.) acute, (ii.) subacute, or (iii.) chronic.

Whatever may be urged on the ground of theoretical pathology, it is certainly difficult to draw any sharp distinction between certain forms of degeneration of the cardiac muscle cells and so-called parenchymatous myocarditis. Profound protoplasmic change in the muscle elements occurs in many toxæmic conditions, and particularly in typhoid, diphtheria, and septic fevers. It is important to remember that a like change occurs in acute rheumatism.

In some cases of "alcoholic heart" the muscle cells present characters indistinguishable from those met with in diphtheria. Influenza has of recent years amply proved itself, in many instances, a serious agent in affecting heart failure. The importance of pneumonia in bringing about rapid cardiac collapse, especially in muscle degraded by chronic alcoholism, is too well known to need more than a passing reference. But so-called parenchymatous myocarditis to a greater or less extent may occur in almost any disease of an infective character, or morbid condition associated with continuous pyrexia.

Special reference may here be made to the "alcoholic heart" so common among heavy beer-drinkers in male labourers, and such as draymen, market-porters, cabmen, and public-house employees, but from which spirit-drinkers are far from being exempt. Although the symptoms of cardiac failure may be of sudden or rapid development, more or less hypertrophy, and often very conspicuous increase in the muscle, is almost always present. The toxic influence of alcohol on muscle leads to degenerative changes, but the enormous quantities of fluid frequently imbibed must not be left out of consideration.

When a myocarditis proceeds to the formation of pus it is spoken of as a suppurative myocarditis, which may be localised, forming one or more abscesses, or more rarely diffuse.

Such results from pyogenic infection. The micro-organisms may reach the myocardium by means of the blood, or by direct extension from the endo- or pericardium. Many are of the same nature as those met with in malignant endocarditis, and produce rapid degeneration and necrosis with a certain amount of inflammatory exudation.

Chronic Interstitial Myocarditis.—Under this heading it has become customary to include those conditions characterised by a formation of fibrous or indurated tissue, when the true muscle elements have undergone wasting or been replaced by sclerotic formation. But myocardial sclerosis is by no means always the result of a true inflammatory process. Hence the above term is hardly sufficiently inclusive to be absolutely accurate.

Arterio-sclerosis of the coronary arteries must be credited with the greater number of cases of fibroid heart. The influence of gout, lead, syphilis, Bright's disease, alcoholism, over-work and old age, in directly leading to such is discussed elsewhere, and calls for no further consideration here.

The myocardial sclerosis may be (i.) local or (ii.) diffuse. When local, and under the influence of arterial supply, the left ventricle usually suffers to the greatest extent. The indurated areas appear as firm, pale, fibrous patches which microscopically present the characters of dense cicatricial-like tissue.

Cardiac Aneurysm.—Local bulging of the myocardium is usually dependent on antecedent disease of the muscle ending in fibroid induration. Exceptionally, however, a cardiac aneurysm forms before fibrous transformation has occurred. The endocardium is also sometimes found bulging between the muscular fasciculi. Cardiac aneurysms are most frequently situated in the walls of the left ventricle, on the anterior aspect and near the apex; but they also arise on the posterior wall and even in the septum, when they may bulge towards or into the right ventricle. In size they vary from that of a marble to a hen's egg, and occasionally may even be of larger dimensions.

Aneurysms in connection with the valves and local bulgings on the coronary arteries are referred to elsewhere.

Hypertrophy and dilatation may occasionally exist separately, but usually occur in combination, constituting the commonest cause of cardiac enlargement. Hypertrophy may always be considered a secondary condition, and is in most instances to be looked upon as compensatory in nature. Dilatation is generally secondary, and mainly indicative of myocardial inadequacy.

Hypertrophy may arise from (i.) intrinsic or intra-cardiac conditions, such as valvular affections, and perhaps sometimes "adherent pericardium," and (ii.) extrinsic or extra-cardiac conditions, such as arterio-sclerosis and chronic Bright's disease. In hypertrophy, consequent on toxic influences, as from alcoholism and excessive smoking, and long-continued neurotic palpitation, as for instance in Graves' disease, probably dilatation is to be considered the first and essential lesion, hypertrophy occurring rather as a later effect. The hypertrophy met with in those employed in laborious occupations, and that occurring in pregnancy, are to be looked upon as in the borderland of the physiological. Hypertrophy is generally to be construed as a reparative or compensatory effort, while dilatation works in the direction of a break in the restorative mechanism except in the case of aortic incompetence, where to a certain degree it is advantageous.

Formerly three varieties of hypertrophy were described—(i.) *simple*, where although the myocardium was increased the cavity remained of normal dimensions; (ii.) *eccentric*, with both walls and cavity increased, the form now better described as "hypertrophy with dilatation"; and (iii.) *concentric*, where the cavity was thought to be diminished, but which is now recognised as due to post-mortem contraction.

The whole heart may be hypertrophied, when it is aptly described as "bovine"; if the right ventricle is chiefly involved a "quadrate" form will be assumed; and when the increase in muscular development predominates in the left ventricle a "conical" heart results, provided, of course, the left ventricle has not become rounded and lost its apex.

It is impossible here to describe in detail what may be termed the local distribution of cardiac hypertrophy. Brief reference can only be made to some few points. The walls of the left ventricle manifest the greatest muscular development, particularly in disease of the aortic valve. In aortic stenosis hypertrophy predominates and may exist alone for a long period, but in incompetence it is associated from the first with dilatation, which, indeed, in a sense may be regarded as compensatory, though hypertrophy must speedily follow. In aneurysm of the thoracic aorta no hypertrophy may occur, provided the aortic valves remain competent. In "granular" kidney enormous and almost pure hypertrophy arises, but even here the right ventricle participates to some extent. Hypertrophy of the left auricle occurs in mitral stenosis. Obstructive diseases of the pulmonary circulation lead to great hypertrophy of the right ventricle, but diseases of the aortic and mitral valves, especially mitral stenosis, are a common cause. In congenital lesions very extensive hypertrophy is often apparent. It may be desirable here to add that even in "right-sided" cases of hypertrophy more or less implication of the left ventricle will also be found, as indeed would be expected when the "solidarity" of the heart is considered.

A hypertrophied heart may assume considerable proportions and increase greatly in weight. The 9 ounces of the normal heart may frequently be doubled, often reaching 25 or 30 ounces, and have been known to touch 46½ ounces. Much discussion has taken place as to the actual change in the muscle elements. It would appear that there is an increase both in the size and number of the cells. *Dilatation* is occasionally detected as a primary condition, but usually occurs in combination with more or less hypertrophy, the latter process tending to stay the former. Sometimes dilatation may occur as an acute condition, as for example in the kidney affections of scarlet fever, but usually it is rather of a chronic and progressive development. While the predisposing causes are many and often obscure, the exciting influences may be readily grouped into two classes: those acting (i.) by diminishing the contractile power of the muscle, and (ii.) by increasing the pressure to be overcome. In the former class may be placed conditions of atrophy and degeneration, some forms of infiltrations, and most inflammatory states of the myocardium. Certain cavities may be more markedly dilated than others, but frequently the whole organ is involved by a general dilatation. When only a part of a chamber is affected it is usual to speak of it as an "aneurysm of the heart."

Symptomatology

The symptoms of heart disease, whatsoever be its form, are very similar, and seeing that they for the most part depend on the same kind of disturbance in the circulation (resulting in venous stasis) of the different organs of the body, it is not surprising that this should be the case. It is on physical examination mainly that we depend, as already stated, for the differentiation of the causes of such disturbance that lie in the heart, but not entirely on this mode of investigation, seeing that it alone will sometimes entirely fail to enable us to form a diagnosis worthy of the name, which attention to the history of the patient will enable us at once to accomplish. This statement will be made clearer by taking two actual cases in illustration:—A man aged forty complains of the following symptoms: shortness of breath on any exertion, dropsy of the lower extremities, and some uneasiness referred to the right hypochondriac region, the first progressive and of six months' duration, the second and third of six weeks' duration. *Signs:* On physical examination the veins of the neck are seen to pulsate while the patient is upright: the heart is found to be somewhat enlarged; over its apex there is a systolic murmur following the first sound, which latter is alone audible to the inferior angle of scapula behind, and the pulse is irregular. Now these conditions, we shall find, are common to two very different forms of heart disease—mitral stenosis and muscle failure. But the *history* of the patient will enable us at once to decide between them, for at the age of fifteen the patient had a severe attack of acute rheumatism and has been a temperate and careful liver. In short, the *symptoms* render cardiac disease probable; the *signs* confirm this opinion, but it is the *history* that enables us to make the diagnosis of mitral stenosis by informing us of the old attack of acute rheumatism which renders that lesion probable under the circumstances. Again, in a man a few years older the symptoms and signs are closely similar, but the history excludes rheumatism and chorea, and proves that his "wind" was good up till the time of his leaving the army six years before, in which he could perform arduous drill with ease to the end, and reveals the fact that since leaving the army he has been a confirmed beer-drinker, and as a consequence has neglected his proper food. The case is one of alcoholic muscle failure of the heart.

But let us suppose that the patient is sixty years of age and had an attack of acute rheumatism at thirty-five, while his cardiac symptoms are of comparatively recent date, say five years' duration, the same physical signs will in the presence of the history leave us somewhat in doubt as to the possibility of there being stenosis as well as senile muscle failure. Still the age at the time of the rheumatism and the recent development of the symptoms of disturbance of the circulation render it probable that the case is one of primary muscle failure, and that the patient escaped any serious consequence of endocarditis. If the patient had aortic incompetence as his lesion, a similar question of pathological diagnosis may arise, and again probability would be in favour of the origin of the disease being degenerative and arising in the vessel, rather than rheumatic and arising in the valves.

As regards symptoms, then, practically all forms of heart disease may be studied together: it is the physical signs they present that accomplish their separation in the light of the past history of the patient and a knowledge of pathology.

There are three symptoms that stand out pre-eminently from among the many to be considered: these are *dyspnœa*, *dropsy*, and *enlarged tender liver*, and they may be regarded as the cardinal symptoms of heart disease.

1. Of the three, *dyspnœa* is probably always the first to manifest itself, though it occasionally happens that patients take no heed of it until dropsy supervenes, which immediately alarms them and renders them observant. In such cases careful inquiry will almost invariably elicit evidence of failure of "wind" on exertion for some time before the appearance of dropsy.

So frequently is this the case that one may be pardoned for doubting the statement of the few patients who obstinately deny having had any dyspnœa before they noticed dropsy. Many people seem to have great difficulty in defining their sensations, and yet give a description of sensations that points strongly to there having been among them the feeling that most people would describe as "shortness of breath" or "breathlessness," without having recognised it as "air-hunger," to use the expressive term of the Germans. It is on *exertion* that the first experience of dyspnœa usually occurs. We shall have, however, to describe certain distressing forms that occur independently of any exertion, but it may be taken for granted that the "wind" of those who suffer from them is always at fault on exertion. *There is no indication of the well-being of a heart to be compared in importance with "goodness of wind."* If the physician is *assured* of this he need hardly examine the heart, for "sound wind" and "an unsound heart" may be regarded as incompatible conditions. Ascent, either of steps or a simple incline, throws a peculiar strain on the heart apparently, and discovers the crippled heart with unfailing certainty. It must be remembered, however, that a man who has followed a sedentary occupation and habitually avoided active exercise, may have "bad wind" without any actual disease of the heart, and simply as the result of his having persistently given the organ the least possible amount of work to do, but such a heart is capable of great improvement under treatment by processes analogous to that employed in athletic training.

A man may have a cardiac lesion—say mitral stenosis or aortic incompetence—and yet suffer no inconvenience in consequence, so long as he is subjected to no strain; but he will not ascend a flight of stairs without some degree of dyspnœa over and above that experienced by the average healthy individual; the effort betrays his weakness, which without such exertion may be entirely latent, thanks to the vigour of the heart muscle.

Breathlessness on slight exertion, then, is the first indication of cardiac failure in general, but in certain cases this is not all, and the patient while at rest in bed during the night is seized —often awakened by—a distressing paroxysm of breathlessness which compels him to sit up and "struggle for breath." This type of dyspnœa is especially apt to occur in cases of Bright's disease and gout—in fact, in cases of muscle failure of the heart in combination with a fairly high degree of arterial tension, and the writer is disposed to believe that associated with such attacks is an abrupt rise of the arterial blood-pressure whereby the heart is seriously embarrassed.

Another form of dyspnœa often noticed in similar cases is that in which the stimulus of volition seems necessary to maintain the activity of the respiratory centre, for when the patient falls off to sleep his respiration ceases, and he becomes cyanotic, often showing in consequence slight convulsive movements of the hands. These are soon arrested on his awaking, gasping for breath. Another remarkable type of dyspnœa that comes on during repose is known as Cheyne-Stokes respiration, in which pauses in respiration occur at definite intervals, each one being followed by the resumption of respiration at first with the shallowest of inspirations, which become gradually both deeper and more frequent until the acme is reached, when the inspirations become less deep as well as less frequent, until another pause sets in, to be followed in turn by another series of respirations of ascending and descending type, and so on. This type of dyspnœa is specially common in the heart failure of chronic Bright's disease (granular kidney). It has also been supposed to be associated with fatty heart. During the paroxysm of dyspnœa patients will sometimes spring out of bed at the maximum of their distress. On the other hand, respiration of "ascending and descending" type may occur without any subjective distress of any kind. A rare and often overlooked form of cardiac dyspnœa, if such it can be called, consists of an occasional very deep inspiration or sigh. Long ago this peculiar sighing respiration was recorded as a sign of "fatty heart." Certainly it belongs to the more obscure cases of cardiac failure, which may terminate by sudden fatal syncope, or which may be revealed by the ordinary symptoms of venous stasis only shortly before the end. The writer cannot say if there has been any stoppage or alteration of respiration immediately preceding these deep inspirations, which occur after long intervals and apparently in the midst of quiet respiration.

2. *Dropsy.*—The second cardinal symptom of heart failure, dropsy, is probably invariably preceded for a longer or shorter period by dyspnœa. It is likely, however, that the tissues through which and into which the exudation of serum takes place undergo some nutritive change, which favours the process. This "tissue-element" in the production of dropsy seems to vary in activity, its working being less apparent in cases of chronic valve lesion, as the rheumatic, and most apparent in the dropsy associated with the muscle failure that results from alcoholism. The tendency to tissue degeneration and vasomotor palsy promoted by this habit is notorious. In the simple rheumatic case the dropsy begins where we naturally expect there being the greatest venous stasis—the lower extremities—and spreads upwards as the conditions that occasion it become intensified. But the dropsy of the alcoholic heart-case is characterised by extraordinary caprice both in its first and later

localisation, and ultimately by its as extraordinary extent of distribution. The first peculiarity is exemplified by such rarities as the scrotum being for a time alone affected, the second by the whole surface of the trunk becoming very dropsical, so that a deep and long-lasting indentation can be made with the finger all over; sometimes the scalp is similarly invaded. In a case of the kind the œdema is sometimes particularly noticeable over the sternum, where a massive pad, pitting an inch or more deep on pressure, is often formed. Over the lower back is a common localisation for dropsy, and this "pitting" over the sacrum may be found actually when there is no dropsy of the lower extremities. This, of course, happens usually in patients who have been recumbent for some time. The frequency of kidney complication towards the end of cardiac cases is very great, and the writer at one time placed this question before himself, Do the upper extremities ever become œdematous in heart cases in which there is no albumin in the urine? and he was soon able to answer the question in the affirmative, though the occurrence is rare. Even dropsy of the face may occur under the same circumstances, though unquestionably it is very much less common than in Bright's disease of the kidneys; in the former case it is never an early dropsy, and is probably always associated with an extensive development of dropsy elsewhere. A comparison of the two dropsies, that dependent on kidney disease and that dependent on heart disease, results in bringing into relief rather their points of resemblance than their points of difference; we exclude from consideration the numerous cases of heart failure secondary to Bright's disease, cases into which a cardiac element is introduced.

Serous exudation into the subcutaneous cellular tissue is very likely to be accompanied by serous effusion into one or more of the serous sacs—pleuræ, pericardium, and peritoneum—but it is often difficult to be certain that a given effusion is a dropsy in the ordinary sense, and not of inflammatory origin. This difficulty is specially met with in the case of pleural effusions, the lung complications in heart disease being so numerous and so apt to set up secondary pleuritic effusion while apparently primary pleuritis is far from rare. Hydrothorax in the sense of simple plural dropsy is generally bilateral, but so may pleuritic effusion be, while simple dropsy may occur on one side only, because the pleural sac on the other side has been obliterated by old adhesions, in which case the lower part of the corresponding lung becomes œdematous. It is a curious fact that ascites is apt not only to be specially pronounced in cases of mitral stenosis, but may actually be the only dropsy present. The circumstance is no doubt associated with the special proneness of the liver to suffer from venous stasis in that disease, and with the long duration of the lesion permitting secondary changes to be developed in the organ.

3. *Enlargement of the Liver*.—This is so marked a feature of the disturbance of the circulation resulting from heart failure that it is fully entitled to be placed with the other two symptoms we have considered as cardinal. Stokes admirably described the condition in 1854.[1] Speaking of an exacerbation in a heart case, he says: "It is under these circumstances that the already enlarged liver exhibits a rapid increase of tumefaction, in a few hours descending far into the abdomen, yet on the subsidence of the attack returning to its ordinary volume, when it may be felt as a flat indolent tumour extending for an inch or more below the false ribs" (p. 257).

When we consider the peculiarities of the portal circulation it is not surprising that the liver should be specially susceptible to the venous stasis resulting from heart failure. That the swelling of the organ, noticeable under the circumstances, is due essentially to the vascular engorgement, is plain not only from the rapid diminution of the organ that can be traced at the bedside when the general circulation is restored, but also from the result of post-mortem examination of patients, who die with the organ engorged, but whose livers collapse on cessation of the circulation. The late Dr. Murchison divided the enlargements of the liver into the *painful* and *painless* varieties, and the enlarged liver of heart disease certainly belongs to the painful variety, but the pain is slight till the organ is pressed upon. Otherwise the organ is smooth, but feels somewhat indurated. This apparent induration (as the tenderness probably) is no doubt accounted for by the tightening of the capsule of the liver as its contents are increased under pressure, for it rapidly diminishes, and often disappears in a few days—in fact as soon as the circulation is restored and the venous stasis relieved. We have incidentally referred to ascites being occasionally the only dropsy present in cases of mitral stenosis,—generally cases of long standing,—and the fact is undoubtedly associated with another fact often observed in cases of the kind, namely, that the liver may be found greatly enlarged and tender on pressure while yet there is not—nor has been—a trace of dropsy of the lower extremities. There can be little doubt that the liver and portal circulation suffer specially in the venous stasis of mitral stenosis, though why this should be so is not clear. *Icterus* is exceedingly common in the later stages of cardiac disease in association with venous engorgement of the liver. The stools, however, usually contain bile. When there is marked cyanosis, as well as jaundice, the

[1] *The Diseases of the Heart and the Aorta.* Dublin, 1854.

patient's countenance is apt to assume a greenish tinge; that to the experienced observer is peculiarly characteristic of the final stage of cardiac disease.

Palpitation, or the too forcible or too frequent beating of the heart, whereby its action becomes perceptible to the individual, is an unimportant symptom in the majority of cases of organic heart disease, although it occupies a high position in the estimation of the laity. Moreover, in some cases of organic disease it is a cause of much distress. The writer has seen this result most frequently in cases of aortic incompetence, in which, notwithstanding huge enlargement of the left ventricle, the right side of the heart was little involved. Also in Graves' disease, which often leads ultimately to cardiac dilatation too, he has witnessed the symptom in most distressing severity. Patients often confuse various disturbances of their cardiac action under the term palpitation, which agree only in one respect, namely, that they make the individual aware of the movement of his heart. Thus the too forcible beat that follows an intermission or an abortive beat, and the peculiar "fluttering" sensation that often accompanies irregular action, are frequently described as "palpitation," so that the physician has to be careful not to accept the term in its proper significance too readily. Sometimes the patient seems to be hyperæsthetic, so that he is aware of his heart's action, though to the observer it seems neither unduly forcible nor sufficiently frequent to occasion the sensation, while it is quite regular.

Cardiac Pain.—The great pain associated with heart disease is angina pectoris, which seems unquestionably dependent—absolutely as far as the writer's own experience goes—on obstruction either at the orifice or in the course of the coronary arteries, one or both. Angina pectoris, however, is specially described in a separate article in this work. (*See* "Angina Pectoris," vol. i. p. 187). But it is astonishing how frequently the subjects of morbus cordis complain of local pain in the region of the affected organ. The writer is aware that the heart itself is stated by physiologists to be directly insensitive to pain, but he has good grounds for questioning the statement in disease. It may be that though in health the heart is insensitive, it acquires sensitiveness to pain in disease. That the heart *itself* does in disease become painful, though not severely, the writer cannot doubt in view of his clinical experience. Such local pain as is complained of by genuine heart sufferers is certainly seldom or never acute and severe, while it is often accompanied by some deep local tenderness over the apex-beat as well as by superficial tenderness to pressure over the second and third left costal cartilages, although the latter is not properly direct tenderness, but is associated with the relationship existing between the visceral and peripheral nerve-distribution proceeding from the spinal segment concerned in cardiac innervation, which is situated in the upper dorsal region.

Pulmonary Conditions.—In correspondence with the early dyspnœa experienced by sufferers from cardiac disease the consequences of lung congestion soon manifest themselves. Among the earliest of these is a hitherto unwonted tendency to bronchial catarrh, with its ordinary symptoms and physical signs: the latter rhonchi and, in severe attacks, moist sounds, which are apt to be most abundant at or to be limited to the bases of the lungs. As such attacks succeed one another these moist sounds linger longer after each attack, until their presence may become habitual, while the rhonchi from time to time clear away. In other cases, with neither present evidence of nor distinct history of bronchitis, moist sounds are found at the bases of the lungs, the bubble they denote being evidently of comparatively small size, while in still other cases a very fine bubbling sound limited to the end of inspiration, to which the term *crepitation* or "vesicular bubble" (Skoda) can hardly be denied, is persistently present for long periods, while rise of temperature, indicative of inflammatory action, is entirely absent. The explanation of these sounds, which are usually heard equally on both sides, is no doubt an œdematous condition of the lung-bases. Nowhere, so well as in the lungs, do we see illustrated the pathological law that venously congested organs are prone to become inflamed.

A large number of sufferers from chronic cardiac disease are carried off in the end by a pneumonic complication, and such processes are often associated with "pulmonary apoplexy," which will be considered in another part of this article (under Embolism).

There is little or nothing distinctive in the sputa of the cardiac sufferer from bronchitis; they present the ordinary characters of "bronchitic" sputa.

Little less important than the lung conditions are affections of the pleura in chronic heart disease. Effusion is usually the most important fact, and such effusion may be of inflammatory origin, or a simple dropsy as that of so many other parts. Pleuritis without effusion is usually secondary to some intra-pulmonary pathological state, such as embolism or pneumonia, when it is of quite subordinate importance. The possibility of one or both pleural sacs having become obliterated as the result of adhesions from old pleuritis must be borne in mind. When such obliteration has taken place, instead of the usual hydrothorax, œdema of the lung occurs. As regards the physical signs of hydrothorax, it is perhaps unnecessary to point out that the fluid in the case of a simple dropsy of the pleura is just as immobile as that of a pleuritis, when the

patient's posture is changed, the old view, that adhesion of the pleural surfaces above the fluid, in the latter case, was the cause of the immobility, being quite untenable. In both cases the dull area corresponding to the fluid presents a convex boundary upwards. Vocal fremitus is always diminished, but by no means always lost. The naturally stronger fremitus on the right side must be remembered. As regards auscultation over the dull area there may be diminution of breath-sounds in all degrees up to silence, or there may be exquisite, though usually not loud, bronchial breath-sound and bronchophony or ægophony. The presence of bronchial breath-sound may be taken to mean that a considerable portion of lung has been rendered airless. It is important to remember, in a case in which there is question of tapping, that a few moist sounds or even friction, when the fluid is of inflammatory origin, must not deter us from exploring, if otherwise the evidence of the presence of fluid is strong. Such adventitious sounds are probably only met with when the effusion is partial, the sounds being produced in the still air-containing lung above the effusion in the one case, and likewise above the effusion in the other case, at a place where the inflamed pleural surfaces are in contact, the friction sound produced between which is readily conducted along the chest wall. The writer has drawn off fluid from a perfectly dull area, over which almost loud friction sound was audible. The accumulation of fluid in the pleura is always an important fact in a heart case, for a moderate effusion may seriously interfere with the already embarrassed pulmonary circulation, and the removal of even such an amount of fluid may afford great relief. On the other hand, just such a moderate effusion of considerable standing may almost suddenly increase, so as to render the whole lung airless. How often is the physician appalled at the post-mortem of a chronic heart case, by finding one pleura full of fluid, the corresponding lung airless, and the other lung œdematous, when he had perhaps only thirty-six hours previously found the fluid in the pleura to reach less than half-way up the chest, and the opposite lung to be free from any serious amount of œdema! This insidious though rapid development of effusion is very often the final pathological event in long-standing cases of chronic heart disease, and it is one of the accidents against which the physician should ever be on guard.

The *hæmoptysis* of heart disease finds its appropriate place of description among the symptoms of the embolic process. That hæmoptysis does occur, however, without the accident of embolism and apparently as a result of the general congestion of the pulmonary circulation only, is almost certain. Of this origin it is seen specially in the young subjects of mitral stenosis, in whom it is not rarely the first symptom that brings the patient under medical supervision.

Kidneys.—With the exception, perhaps, of the lung, in no organ are the changes that result from venous stasis of greater practical importance than in the kidney. In the great majority of cases of heart disease there is *albuminuria*, and in the majority of cases with albuminuria the pathologist would report that the kidneys were only "venously congested"; in a certain number he would find evidence of long-standing chronic disease of the kidney (granular kidney most commonly), while in the rest he would pronounce the organs to have become recently inflamed, and to present the macroscopical appearances of a tubular or "mixed" nephritis. Clinically, the difficulty of distinguishing between these pathological states is often very great indeed. The writer remembers an old man, suffering from general anasarca, engorged liver, and pulmonary apoplexy, the results of muscle failure of his heart, whose urine was high-coloured, deposited deep-coloured urates, and had a specific gravity well over 1020, and yet contained a mere trace of albumin. Surely under the circumstances a very much larger quantity of albumin would have been no necessary indication of kidney disease? Yet this patient had exquisitely granular and contracted kidneys. The abundance of albumin, on the other hand, that is quite commonly present towards the end of heart cases, without there being any macroscopical nephritis, is notorious. The mere presence of casts will not decide the question whether we have to do with a "congested" or an inflammatory or a degenerate kidney, although abundance of casts, especially other than hyaline, points strongly to there being more than mere congestion of the kidney. As to the nature of the casts, apart from their number, hyaline and slightly granular casts are most common in cases of the simply congested kidney, while many epithelial and leucocyte casts point decidedly to actual nephritis. In long-standing cases of granular disease of the kidney with secondary muscle failure of the heart, it is, however, often extremely difficult to find casts in the urine at all, because there are so few present. Thus to determine the presence of actual kidney disease in cases of heart failure, in one form or another, may be a practically impossible task. Occasionally the establishment of the diagnosis of kidney disease is at once attained, not by any examination of the urine, but by the use of the ophthalmoscope, "albuminuric retinitis" being so revealed. The urine of pure heart cases is generally high-coloured—apart from the presence of bile—the character no doubt proceeding from the congested state of the liver as well as being promoted by the concentration of the secretion. A persistently pale and comparatively scanty urine is, again, suggestive of

advanced kidney disease, though under the circumstances it is not likely that cardiac failure will be pronounced. The writer is not aware that those who have devoted special attention to urinary casts can assert from the kind of casts present in a heart case that the limit of mere "venous congestion" has been passed, and that true nephritis is established. No doubt abundance of casts, and specially of casts other than hyaline, which have least value, points strongly to the passage having been made, or to there having been *old* nephritis. The frequent occurrence of embolism of the kidney in heart disease must be borne in mind with regard to albuminuria, and especially hæmaturia. In one form of heart disease hæmaturia is specially frequent—not a transient appearance of blood, as in embolism, but one usually persistent till the end of the case—in malignant endocarditis.

The *spleen* suffers little from venous stasis in cardiac disease, evidently because of the ready distensibility of the liver, which is interposed between it and the obstruction. Clinically it seldom happens that the spleen is found considerably enlarged from this cause, and when it is the liver has probably undergone some secondary change, impeding the circulation *in situ*. When the primary obstruction is situated in the liver, as in interstitial hepatitis, on the other hand, enlargement of the spleen is comparatively common. When the spleen is found markedly enlarged in a heart case the occurrence is so unusual that special circumstances are at once suggested: either there has been recent large infarction of the organ, or there is septic endocarditis, which is associated with enlargement of the spleen in the same way as enteric fever is similarly associated, and the enlargement of the spleen is thus not the result of mechanical interference with its circulation—of venous stasis. Whenever the enlargement is considerable the lower end of the organ can be easily felt below the costal arch, while its descent downwards and towards the right, with inspiration, is easily ascertained.

Stomach Symptoms.—The stomach in heart failure being specially subjected, like the other organs in the distribution of the portal circulation, to venous stasis, becomes prone to suffer from catarrhal inflammation of its mucous membrane. Attacks of catarrh vary in their acuteness, and in some cases a subacute or chronic condition is assumed, generally the result of faulty treatment, and specially in the matter of feeding. It often happens that a patient with advanced heart disease has been by habit a good feeder, and may remain so even up to the end. The writer well remembers how the fact struck him, coming to a general hospital after two years' work in a fever hospital, the association of intense illness and the ability to eat and enjoy a meal being of course utterly unknown in fever practice. When there is no gastric catarrh it is not a rare event to see a patient, in the last stage of heart disease and not far from being *in extremis*, eating apparently with relish an ordinary meal. But even in such a case there have usually been attacks of sickness and vomiting during which there has been positive loathing of food. In these attacks the tongue usually becomes thickly coated, and there is often a peculiar odour in the breath—suggestive of that of chloroform—while the urine will often show a port-wine colour on the addition of the liq. of the perchloride of iron. This coloration is apt to be interfered with by the precipitation of phosphate of iron, which dissolves on further acidulation. This "acetone" odour in the breath—sometimes in the urine—of a patient with heart disease leads the expert observer at once to seek for other evidences of gastric catarrh. It is possible that such attacks are often immediately set up by inappropriate and irritating food. In a case of the writer's—an advanced one of mitral stenosis—the patient, a girl, had surreptitiously obtained an orange from her neighbour, and seeing the nurse approaching, gulped down the greater part of it in a mass, which was found post-mortem in her stomach seventy-two hours later, the symptoms of a most acute gastric catarrh having supervened, and speedily brought about a fatal termination. Even in this place the writer would call attention not only to the uselessness, but the actual harmfulness, of continuing the attempt to feed the patient by the mouth during severe gastric catarrh accompanied by vomiting. On the other hand, if the stomach is subjected to no medicinal treatment, but is simply allowed to rest absolutely—what feeding is necessary being accomplished per rectum—recovery is usually speedy.

Among the clinical symptoms of angina pectoris the eructation of flatus towards the end of the attack has long been noticed, and there seems to be a curious association between eructation of "wind" and unmistakable cardiac suffering. So much is this the case, that in questioning uneducated patients, the subjects of heart disease, with regard to their "wind" in the athletic sense of the word, they often misapprehend the application of the term, and enter into a lengthy description of their suffering from abdominal distension and the relief obtained when they can belch up the peccant "wind." The frequent association of the two conditions—heart disease in some form and abdominal distension with "wind"—at least the feeling of it—is too common to be ignored, whatever be its explanation. The subject is one of considerable interest with regard to diet in heart disease.

The intestines are no doubt apt to suffer from catarrhal conditions in a manner similar to the stomach, but it has become so much a custom

to give at least occasional purgatives in the treatment of heart disease, that the influence of these in the production of intestinal catarrh has to be kept in mind. Apart from the use of purgatives, constipation is, perhaps, a more commonly troublesome symptom than looseness of the bowels. It often happens that, while there is habitual constipation, diarrhœa sets in from time to time, probably the result of irritating substances being formed in the intestinal tract. Here, as in the case of the stomach, the importance of the manner of alimentation of the cardiac sufferer is immense.

Hæmorrhage from the Portal System.—In severe gastric catarrh a little blood may appear in the vomited matters, but such hæmorrhage is usually very scanty. It occasionally happens, however, that a profuse bleeding occurs in a heart case per rectum, and is preceded by pain and a tendency towards collapse. In the worst of such cases embolism or thrombosis of large mesenteric arteries is the likely lesion to be found post-mortem, but more than once the writer has known profuse hæmorrhage from the bowels to occur in heart disease without there being found post-mortem any lesion to account for it.

Cerebral Symptoms.—Towards the end of cardiac cases mental disturbance is not uncommon, and assumes different forms, no doubt determined by the hereditary and personal predisposition of the individual. The writer doubts if any special form of mental derangement can be definitely ascribed to cardiac disease, much less to special forms of it. Painful delusions of suspicion are common, and under their influence patients will rise from their beds and subject themselves to efforts they might be thought incapable of. It happens as a consequence that a patient by thus throwing a strain upon his enfeebled heart may render impossible further rally, though the case is often nearing its end before the supervention of mental derangement. The many forms of heart disease, and the multifarious associations of the disease, must introduce toxic and other conditions as capable as the disturbed circulation of disordering the functions of the brain. For instance, the extreme cyanosis accompanying the cardiac failure of pulmonary emphysema is often associated with bad dreams, in which the patient vociferates loudly, so that in a ward he becomes an unmanageable nuisance to his neighbours. In the heart failure of Bright's disease, again, toxic conditions, determined by the kidney disease, probably have much to do with the mental disturbance, which resembles rather that of the "typhoid state" in general, than that associated with venous stasis. Active delirium, again, is a common accompaniment of the hyperpyrexia of acute rheumatism, so apt to be associated with endo- and pericarditis. Perhaps the most definite association, as cause and effect, of heart disease and cerebral disturbance is the hemiplegia resulting from cerebral embolism, which will be considered later.

Epileptoid and syncopal seizures are common as the result of sluggish cerebral circulation in cases of bradycardia. In extreme aortic incompetence mental disturbance is perhaps specially common, and in its production the wide variation between the maximum and minimum blood-pressure may play a part. The cyanosed, bloated face of the sufferers from extreme mitral stenosis or the cardiac failure of pulmonary emphysema contrasts strongly with the pale face of the subject of free aortic incompetence, and it seems reasonable to suppose that the two physiognomies reflect to some extent the mode of interference with the cerebral circulation, notwithstanding its peculiarities.

Active mental derangement is too often a lethal indication in cardiac cases, and the fact is to be explained by this derangement being a late symptom, and therefore likely to be associated with the more lethal conditions of other important organs, including the heart itself, and specially by the very grave opposition to the treatment of the case it offers. Thus if a patient in the late stages of cardiac disease insists upon going down and up stairs on his own legs, the effort may prove speedily fatal to the already over-taxed heart, and the medical attendant has no choice except between heavily narcotising him (not always a safe and easy thing to do) and restraining him by the hands of attendants. Strapping any patient down in bed is a relic of the barbarous ages that has hardly quite gone, but to strap down a cardiac case would simply mean manslaughter, so certainly and speedily would it determine hypostatic congestion of the lungs. After a prolonged forced sleep it often happens that the patient wakes up, if not *compos mentis*, at least amenable to the care of his attendants, but occasionally the morbid activities themselves awake likewise as if refreshed by rest.

Thrombosis of large veins is an occasional result of cardiac disease producing in the lower extremities the conditions so well known as "Phlegmasia alba dolens" (though it is not always painful), liable to occur after parturition, fevers, etc. In heart disease the accident is to be regarded as of very unfavourable prognosis, not so much because of its intrinsic gravity as of the state of general circulation and vitality it denotes. Moreover, the possible detachment of particles of clot and consequent pulmonary embolism has to be borne in mind. The dropsy that results from it is distinguished from cardiac dropsy by its being usually limited to one extremity, or if involving both extremities, by its being greater on one side than the other. Cardiac dropsy is, however, not invariably symmetrical, and the cause of such asymmetry is not always clear. It is obvious, however, that in a cardiac case ordinary dropsy is likely to be

present, which the dropsy of the thrombosis only complicates. The patient's complaining of pain at the seat of thrombosis will often lead to the discovery of the accident, and when a cardiac sufferer complains of pain in a limb it is always necessary that the vessels—both veins and arteries—should receive careful physical examination for vascular obstruction. A more or less elongated mass or cord may easily be felt in some cases, while in others the seat of the obstruction has to be inferred from local tenderness and the disposition of the dropsy.

The Embolic Process

The plugging of peripheral vessels in the various organs by particles, usually of fibrin, from the cardiac cavities or from diseased surfaces of valves, plays an exceedingly important part in the symptomatology of cardiac disease. The accident, as it may be called, of embolism probably always implies either stasis of blood in one or other of the cavities of the heart, as a result of which, fibrin is apt to be deposited from the blood in some of the recesses of the cardiac wall, or the exposure of some abnormal and devitalised endocardial surface, usually of the valves, to the blood-current, whereby fibrin is deposited, as happens in the case of rheumatic and septic endocarditis. *Cerebral embolism* is a common accident in mitral stenosis, and the source of the plug is usually a tiny clot detached from a recess in the wall of the left auricle. But cerebral embolism may occur also in cases of simple dilatation of the heart, the source being either the left ventricle or auricle, in either case presumably in a state of "systole catalectic," so-called "asystole." The process is perhaps most common in the lungs, in which case the source of the peccant particle is the recesses of one or other of the right chambers. The symptoms of embolism will be considered according to the organ affected.

Pulmonary Embolism.—The so-called "*hæmorrhagic infarct*" described under pathology is the lesion here,—"pulmonary apoplexy" is the old-fashioned name for the condition, an inappropriate one, however, as regards its etymology. The great *symptom* of the condition is *hæmoptysis.* The blood is generally pure, dark, and non-frothy, and the quantity varies much, but it never becomes so great as to necessitate the mere loss of blood being taken into consideration. With or preceding by a short interval the appearance of hæmoptysis, there is often some febrile disturbance, which, however, is usually neither great nor prolonged. On physical examination it is the rule to find no marked dulness over the site of infarction, the patch being usually small and isolated, but when there are several infarcts lying close together, and especially when inflammatory consolidation takes place around infarction, a considerable area of dulness may be produced. The most common auscultatory sign is the development of small bubbling sounds, the bubbles formed being, however, of a larger size than those that occasion the "vesicular bubbling" of Skoda or *true crepitation,* such as is heard in the incipient stage of pneumonia and in œdema of the lung substance. Bronchial breathing is exceptional, the breath-sound being usually "indeterminate" (Skoda) or essentially vesicular. Bronchial breathing implies, of course, that a large mass of consolidation has been produced, which is not often the case, and that the large bronchial tubes of the consolidated part are free for the passage downwards of the glottic breath-sound—a condition, one would think, often prevented by the presence of blood in them. The physical signs in typical cases are developed over a comparatively small area corresponding to the localisation of the infarct—a subject considered under pathology. When a large area, say the greater part of a lobe, is rendered dull, the presumption is that ordinary inflammatory consolidation has supervened around the infarct or infarcts, and the temperature is then apt to be maintained at a high elevation. When a large embolism occurs or several small ones occur in close proximity, pleuritic effusion is apt to result and mask the primary condition, although hæmoptysis will usually be present to indicate that the embolic process is in operation.

Cerebral Embolism.—A very important part in the symptomatology of cardiac disease is played by the embolic process involving the brain. Among the vessels, in which the embolus may be arrested, the middle cerebral artery of the left side stands pre-eminent. Its occlusion gives rise to right hemiplegia and usually more or less aphasia, the latter being sometimes very complete. Differences among cases depend essentially on the exact site of the lesion, and consequently the branches whose circulation is interrupted by the embolus. The "simple" mode of onset of hemiplegia, in which there is no loss of consciousness, is the rule, though occasionally the "epileptiform" and "apoplectiform" modes of onset are witnessed. The attack is remarkable among "strokes" in general, because of its frequent occurrence in the young; so much, indeed, is this the case that a "stroke" in a young person, especially of the female sex, at once suggests heart disease, and specially valve disease. Quite a large proportion of young female patients suffering from mitral stenosis become the subjects of such attacks of hemiplegia. The form of hemiplegia is not always the same, and the left side—the embolus being lodged in the right cerebral hemisphere—may be the affected one, as in a young girl lately under the writer's care. Moreover, owing to unusual vessels becoming plugged, instead of a definite hemiplegia resulting, quite irregular and anomalous manifestations of locally arrested cerebral circulation may arise, and these may

be either trivial or grave and lethal. Thus the writer, when as a house-physician making one evening his usual round, while actually feeling the pulse of a patient suffering from cardiac disease, noticed the hand and forearm twitch convulsively, and the patient bcame suddenly sick, and immediately complained of violent headache. Next day there was still headache, and the head could not be raised from the pillow without sickness. Slight paresis of the left 6th nerve was noticed on the following day, but all the symptoms of the seizure had gone, and the ocular paresis disappeared a few days later. A fortnight after the same patient died from an embolus which evidently blocked a large vessel, violent headache in the occipital region, urgent vomiting, and speedy loss of consciousness being the symptoms produced, death resulting two hours after their sudden onset. Between these extremes all degrees of severity of seizure may be witnessed as the result of cerebral embolism, but as the special symptoms of the embolic process affecting the brain are considered in detail elsewhere in this work it is useless to refer to them at greater length here. Aneurysm or rupture (with fatal apoplexy) may be final results of embolism of cerebral vessels, and in regard to them the reader is referred to the section on pathology.

The writer has had no clinical experience of arterial embolism in the liver, while the condition of this organ generally in heart disease has been fully considered.

Embolism of the spleen produces occasionally swelling of the organ, accompanied by some degree of pyrexia and local discomfort or actual pain. When the peritoneal covering becomes inflamed the pain may be acute, and a friction rub may be detected by stethoscope and hand. The clinical detection of splenic embolism is only likely to be accomplished when the infarction is large or multiple.

Embolism of the mesenteric arteries is accompanied, when the vessel blocked is large, by sudden abdominal pain followed by the indications of shock or collapse, and the later occurrence of profuse hæmorrhage per rectum. The accident is a rare one, and the writer has observed only two cases, in one of which the patient recovered. In the other the condition was proved post-mortem.

Large peripheral arteries—as the brachial, femoral, and popliteal vessels—are occasionally the seat of embolism, severe local pain ushering in the attack, and being followed by numbness and paresis of the limb, which becomes cold, pale, and finally livid. Aneurysm may ultimately result at the site of obstruction, owing to the necessarily impaired nutrition of the vascular wall at this spot.

There is usually little difficulty in finding the exact situation of a peripheral arterial plug. Gangrene of the part supplied may result, but in most cases a collateral circulation is easily established, the arteries of the part being sound.

In the foregoing account of the symptoms of cardiac disease in general, these are regarded as the product of that condition of circulation which we have designated "venous stasis," characterised by excess of blood-pressure on the venous or "return" side of the circulation. It must be admitted, however, that clinical experience makes us acquainted with the fact that heart cases are met with in which "venous stasis" is conspicuous by its absence, to use the hackneyed phrase, and this to the end of the case. Of the three cardinal symptoms the first —dyspnœa—under these circumstances, the writer believes, is never altogether absent. The sedentary man with failing heart, however, may never be aware of his disability, because of his never putting his heart to the test before sudden fatal syncope occurs. The writer well remembers the sudden fatal syncope of a relative of his own, who had never manifested any of the ordinary symptoms of cardiac disability till instantaneously fatal syncope occurred utterly unexpectedly in the midst of tranquil occupation. Yet the writer had often noticed as a boy this same relative give now and again that peculiar sigh he has described as a rare form of dyspnœa. Again, in the case of a friend, while apparently in perfect health, he often noticed this same occasional sigh for years before more ordinary symptoms of cardiac failure made their appearance, and the subject of them died in middle life after a short illness in which neither murmur nor dropsy was present, and in which the expression of the cardiac failure appeared on the arterial rather than on the venous side of the circulation, if one may so say. But even such cases bear out the belief in the paramount importance of respiratory interference in some form in all cases of cardiac failure. The writer has no doubt whatsoever that had the individuals whose cases he has referred to been subjected to effort their "wind" would have failed in the ordinary manner of cardiac dyspnœa. While admitting, as he must, the occurrence of such unusual cases as those related, he is firmly of opinion that had the patients survived longer venous stasis with its ordinary manifestations would have supervened. This conviction is derived from his observation of patients who first of all developed the symptoms of failure on the arterial side of the circulation, but later—often, indeed, only shortly before the end—the ordinary ones on its venous side. The two conditions that are specially apt to furnish examples of failure on the arterial side are aortic incompetence and fatty heart. The consideration of angina pectoris is excluded from this article. The curious condition known as bradycardia, which is usually associated with degeneration of the cardiac muscle, but which may complicate valve lesions, as mitral stenosis, may be regarded

as a third. The comparatively slow pulse of aortic stenosis probably belongs to a different category, and within limits may be a physiological adaptation. Congenital bradycardia, too, must be regarded apart. Failure of the cerebral circulation is the striking feature of these rare cases in which manifestations occur on the arterial side—syncope, epilepsy, and distressing "cerebral" sickness being common among them. These symptoms are best developed in cases of simple muscle failure. Patients with aortic incompetence, again, may have extremely developed physical signs of their disease, and suffer much from dyspnœa and palpitation, without any development of dropsy or engorged liver, and they may go on in this condition for years. But even when this has been the case, the two lacking cardinal symptoms may become developed with great rapidity and in great intensity during the last few days of life. Between these two conditions an intermediate one may be quite unexpectedly produced, the features of which will be best described by an illustration. A man in early middle life, who had free aortic regurgitation, the result apparently of strain, and suffered severely from dyspnœa on exertion and anginal seizures on any effort, though he had never had dropsy or engorged liver, went to his work as usual one morning. Soon after he had commenced his work severe dyspnœa set in, and in spite of rest and careful treatment from the outset of the attack (it occurred in hospital, the patient being an employé), became more and more distressing, while signs of congestion of the lungs developed, with physical signs, for the first time, and the patient died towards evening, the post-mortem revealing, besides the aortic lesion and a dilated left ventricle, intense congestion of the lungs. Who can doubt that had this patient survived longer the lacking cardinal symptoms—dropsy and engorged liver—would have been speedily produced, and thus the case would have been brought into the ordinary category of chronic heart disease? The writer, in a case of aortic incompetence, was once struck, on feeling the patient's pulse, with the extraordinary length of intermissions that were recurring from time to time, and a few hours later the patient quite suddenly died, without any noticeable alteration of his condition. Can we doubt that an intermission, during which the relaxed ventricle would be exposed to the forcible rush of blood resulting from arterial recoil, overbalanced itself, so to speak, in such a way that it was unable to recover? And if this may happen in aortic incompetence, may it not also happen in cases of simple muscle failure, in which an intermission has tarried too long? Nay, even in the healthy heart may not the physiological intermission of cerebral inhibition occasionally pass beyond the point from which recovery can take place?

Lastly, the justification for the old view of sudden death from spasm of the heart must be considered. After death in cases of the kind the cavities of the ventricles are found obliterated, except the small supra-papillary space which contains blood. This occurrence cannot, however, be definitely associated with sudden death, after which flaccid ventricles are commonly found, and it may be that the tetanic contraction of the ventricles occurs only after their paralysis has brought the circulation to a standstill and the organism to somatic death. The one condition is quite as lethal as the other, and it is obvious that their effects will be practically the same as far as the circulation is concerned.

It is a well-known fact that, in a disease usually regarded with so little apprehension as anæmia, sudden fatal syncope may occur. Nay, even when the heart, as far as we know, is in perfect health, profound nervous impressions may induce fatal syncope, for the cause of which the most skilled and careful pathologist will look in vain, and such cases have occurred in young subjects. In most cases, however, of *apparently* appallingly sudden death, clear indications of cardiac failure have been present for a considerable time, though probably known only to the patient, who, ignorant of their significance, gave them no heed. When a man drops down dead in an assembly or in the street, it will often be that the transition from health to death has really been a very much longer process than it often is in pneumonia or malignant scarlet fever. It is, indeed, the latency of the disease rather than the suddenness of its termination that is remarkable. Rupture of the walls of a flabby heart or of a cardiac aneurysm are of course well-known causes of sudden fatal syncope, and it may be well to note that convulsion, from suddenly produced cerebral anæmia, is a common manifestation of the sudden failure of the heart's action in cases of the kind. Angina pectoris, with which we have nothing to do here, is a subject closely related with that of sudden fatal syncope, and in the writer's belief has its anatomical basis in disease obstructing the coronary vessels.

These brief remarks must suffice to indicate the few cases of heart disease that prove fatal without the development of venous stasis, a condition which would seem only to have been anticipated, and thus to have been given no opportunity for development.

A note may here be made of rare cases of mitral stenosis that prove fatal from bronchitis and engorgement of the lungs without the development of dropsy. The liver under the circumstances usually affords indications of venous stasis.

Malignant Endocarditis

A short account must be given of the sym-

ptomatology of septic endocarditis.[1] This remarkable disease presents itself to clinical observation with symptoms belonging to an entirely different category from that to which the symptoms later described belong. The element of *venous stasis* we found to predominate in the production of most of the latter, and for those that remained the *embolic process* was largely responsible. But septic endocarditis commonly presents itself under a clinical aspect that bears little or no resemblance to that of ordinary heart cases. Constitutional disturbance of the usual febrile type is early manifested, and generally brings the patient to rest in his bed before any marked degree of even the first of the cardinal symptoms has developed. It is for such febrile suffering that the patient usually first of all seeks advice, and the evidence of cardiac disease is discovered only in the course of thorough systematic physical examination. The pronounced anæmia that is often early developed in the disease may indeed lead to even such physical evidence as there is being ignored—for instance, to the murmur of mitral or tricuspid incompetence being altogether attributed to the muscle failure of anæmia. This could only happen to the grossly careless observer, who remained ignorant of the daily rise of temperature, not to mention other symptoms. The spleen, as already mentioned, is enlarged in the great majority of cases of septic endocarditis, while it is only exceptionally so in ordinary heart cases.

"Typhoid" symptoms—dry tongue, delirium, subsultus, etc.—may be developed, but it is often surprising how long they remain absent in the presence of prolonged intermittent, remittent, or irregular pyrexia, the patient retaining a fairly clean, moist tongue, and some appetite, while his mind is entirely unclouded for months,—the writer has known a case last over a year,—in these respects the case resembling a tuberculous one. Albuminuria and hæmaturia, and the presence of casts in the urine, are of frequent occurrence. Petechiæ, with the development in some degree of a hæmorrhagic diathesis, are not rare. Hæmorrhages into the retina are apt to occur, and even optic neuritis has been observed on ophthalmoscopic examination. The occurrence of embolism in various organs is common, and the corresponding symptoms, so produced, help to make up the clinical picture of the disease, which latter, in spite of the views of pathologists as to its being capable of production by different microorganisms, forms a distinct and, on the whole, well-defined clinical entity usually easy of recognition when its broad features are known. Aneurysms may develop at the seat of embolism and even in the case of large arteries—for instance, the femoral artery, as in one of the first cases seen by the writer. The cerebral hæmorrhage with apoplexy, that occasionally proves fatal to quite young subjects of the disease, is probably likewise associated with the embolic process.

[1] For Etiology and Pathology, see p. 91.

In a large proportion of cases septic endocarditis complicates cases of ordinary chronic valve lesion—generally rheumatic—when the symptoms referred to become added on to those of such chronic valve lesion.

It has become customary to describe certain clinical types of the disease, although the advantage of such attempted classification is open to question.

(1) The septic type is characterised by the occurrence of rigors and perspirations, the "typhoid" state, occasionally more or less icterus, multiple arthritis, with or without pus formation, and hæmorrhagic and erythematous eruptions.

(2) The "typhoid" type resembles in its symptoms enteric fever with tympanites and diarrhœa, delirium, somnolence, or coma. In relation to this type the occurrence of infarcts in the intestines and of occasional ulceration of the bowels is noteworthy. It must be remembered that murmurs (systolic) arise not very rarely in cases of enteric fever independently of any endocarditis, and are probably associated with muscle failure of the heart.

(3) The "cardiac" type is that which usually has been preceded by rheumatic endocarditis and the lesions resulting therefrom. Indications of disturbed general circulation are more likely to be present than in other forms. In certain cases the two forms of endocarditis are difficult to differentiate. The presence of fever, especially in the evening, without the occurrence of arthritis, and otherwise unexplained, should suggest the possibility of the supervention of the septic disease. Enlargement of the spleen will increase apprehension in this direction, but the spleen is not enlarged in all cases of septic endocarditis, and may be temporarily enlarged from infarct in non-septic cases.

(4) The cerebral type is often associated with meningitis—cerebral or cerebro-spinal—but, as in the specific fevers, severe cerebral symptoms are apt to arise independently of gross lesion in the brain. A "comatose" form of septicæmia, apart from any endocarditic implication for instance, is well known (*vide* Fagge, vol. i. p. 587). Embolic hemiplegia and apoplexy from cerebral hæmorrhage, which is probably associated with the embolic process, form another "cerebral type."

With regard to *diagnosis* the septic endocarditis is specially apt to be confused with enteric fever, the two diseases having many symptoms in common. Widal's typhoid reaction now proves of the greatest value in distinguishing between them. The presence of hæmorrhages in the retina would afford evidence in favour of septic endocarditis.

Diagnosis. — A bacteriological examination, unfortunately, in this disease is usually of but little diagnostic help. Still, in every case, it is most desirable that a thorough investigation of the blood should be made.

1. As pointed out in the pathological portion of our article, *leucocytosis* is generally well marked in cases of malignant endocarditis. This may prove of some diagnostic service in certain cases.

2. Many investigations have been made by numerous observers respecting the bacteriological characters of the blood, but with rather meagre results.

The blood is best obtained by the withdrawal with a hypodermic needle and syringe (used, of course, with the strictest antiseptic precautions) of some 5 to 10 c.c. from one of the superficial veins of the arm. This is then mixed with a suitably prepared and liquefied agar-agar culture medium, poured into Petri's dishes, and allowed to develop under proper conditions. By such an examination streptococci and staphylococci have occasionally been found. This method, however, has not proved satisfactory for diagnostic purposes in most cases, as usually the organisms are not freely circulating in the blood, and even when they are exceptionally met with occur in very small numbers.

3. It is interesting also to remember that Widal's reaction has been obtained in cases which have ultimately been found to be associated with the evidences of a malignant endocarditis, probably developing during typhoid fever, or possibly due to the bacillus typhosus.

Special Treatment.—Much of what is later said regarding the general management of cardiac affections is fully applicable to cases of malignant endocarditis.

Since, however, the disease is dependent on microbial invasion various measures have been suggested with a view (*a*) to destroy the organisms; (*b*) to lessen or annul the influence of their toxic products.

No medicinal agent is known which has been proved to directly destroy or arrest the development of the invading organisms. Various antiseptic and disinfectant drugs have been advocated, but their utility is very doubtful. Quinine, sulpho-carbolates, benzoates, salol and mercurial preparations have been used, and according to some with benefit in certain cases.

Arsenic has been strongly recommended, and is sometimes advantageously combined with quinine.

In a few cases hypodermic injections of pure yeast ferment have been administered.

Nuclein and nucleinic acids have also been advised.

All such agents, unfortunately, have proved quite inadequate in the majority of cases, and their use is not encouraged by what is known of the bacteriology of the disease.

The success of serum-therapy, however, in certain of the well-defined infective processes has suggested the application of such to malignant endocarditis. Hitherto the treatment has not always been conducted with the necessary scientific precision, and up to the present time the success has been very limited. Before any antitoxin can be administered with anything like bacteriological accuracy the exciting organisms must be detected. As indicated in the pathological section of this article, various different organisms are capable of setting up the conditions included under "malignant endocarditis." Unfortunately, in most instances, examination of the blood gives no help in ascertaining the particular organism present in the valves. Sometimes, however, when the primary infecting focus is known, or when an investigation of the infecting channel gives a strong indication of the probable organisms responsible for the endocarditic process, administration of the special antitoxin may not only be considered justifiable, but may prove serviceable. According to the nature of the specific irritant, antistreptococcic, antistaphylococcic, or antipneumococcic serums may be employed.

The possibility of mixed infection must be borne in mind, so that, for instance, if a case following pneumonia is not benefited by anti-pneumonic serum the administration of Marmorek's antistreptococcic serum may be justifiably tried.

Physical Examination of the Heart

The Physical Methods of Diagnosis.—*Inspection.*— In a suspected cardiac case the first point to be noted is the vascular condition of the neck. In nearly every cardiac case some evidence of abnormality in the circulation will be obtained on examination of this part: there will be exaggerated arterial pulsation, or there will be venous pulsation, while in aneurysm of the aorta, with aortic incompetence, there will likely be tracheal tugging when the chin is elevated. In many individuals free from cardiac disease in the ordinary sense there is some venous pulsation in the neck in the recumbent posture. On the other hand, there are certain peculiarly short-necked, stout individuals who even when suffering from cardiac disease give little evidence of visible pulsation in the neck, presumably because their vessels are so well hidden. In most cardiac cases, with enlargement of the left ventricle, the carotid pulsation is exaggerated in some degree so as to form a noticeable feature of the neck. It is, however, in cases of aortic incompetence that the visibleness of arterial pulsation reaches its greatest development, because in this disease there is the greatest variation between the maximum and minimum blood tension in the arteries. Increased visibleness of pulsation in the carotid

is best observed in the upper part of the neck towards the angle of the jaw, while pulsation in the veins is best seen at the lower part of the neck. Venous pulsation in the neck while the patient's body is upright may be regarded as always abnormal, but it must be confessed that the circulatory disturbances need not be serious. For instance, most cases of chlorosis or anæmia show it. The pulsation is seen to be double, and a tracing of such pulsation taken with Dr. Mackenzie's phlebograph shows that there is indeed a double wave—a small and a large one. Till Dr. Mackenzie demonstrated the error of previous interpretations, it had been assumed that the small wave was the auricular wave, and the large wave the ventricular, while in reality the opposite is the case. A moment's consideration will show that Dr. Mackenzie's interpretation is the right one, apart from actual observation with the aid of instruments, by which the venous and arterial pulses of the patient are recorded simultaneously on a revolving cylinder. When we consider the relation of parts, the vein that is visible in the neck, the auricle and the ventricle, realise that the contraction of the auricle is immediately succeeded by the contraction of the ventricle, and remember that between the vein and the ventricle the auricle is interposed, we have only to ask ourselves the question: What is happening in the auricle during the first part of the ventricular systole? to be convinced that in the cervical vein during the first part of ventricular systole there must be a negative, not a positive wave, inasmuch as between the contracting right ventricle and the visible cervical vein is interposed the *expanding* right auricle, for immediately its contraction is over this last must expand and give rise to a negative wave in the veins of the neck. Not until the chamber has become again full will its contents transmit to the veins of the neck the ventricular impulse. It does happen in advanced disease of the heart that the ventricular contraction may be manifested by a positive wave in the veins of the neck from the outset of systole, but before this can occur the contractile power of the auricular walls must have ceased, and the chamber have become as it were a passive reservoir. The great majority of visible venous pulsations are double and of the auricular type described, although the venous pulse *may*, as stated, be alone represented by a ventricular wave occupying the whole period of the systole of the right ventricle. It is a remarkable fact that venous pulsation in the neck does not always coincide in frequency with the arterial pulsation: *extra* venous impulses being, as it were, interpolated from time to time. This was well seen in a case of bradycardia under the writer's care.

In many cardiac cases the most pronounced visible pulsation in the neck is that of the vein, as is often observed in mitral stenosis. In aortic incompetence venous pulsation may be absent, while arterial pulsation is apt to be exaggerated in an extraordinary degree. When in the latter disease, however, both kinds of pulsation are present, the inference is that the lesion on the left side of the heart has seriously interfered with the pulmonary circulation and led to much disturbance in the function of the right chambers. Sometimes other veins than those of the neck, as those of the chest, face, and upper extremities, are seen to pulsate, and the liver may pulsate in an expansile manner. Tracheal tugging is a sign of aneurysm of the aorta, and cannot be regarded as a sign of heart disease, though aneurysm frequently produces incompetence of the aortic valves: hence the necessity of reference to the sign here. Apart from true aneurysm, the arch of the aorta may be dilated, and thus its impulse be exaggerated, so as to be perceptible in the supra-sternal notch and at the sternal end of the right 2nd intercostal space. Deformity of the chest, however, may be the sole cause of such abnormal pulsations.

Capillary Pulsation.—In cases of aortic incompetence, on making a patch of erythema by rubbing the nail over the forehead, this phenomenon is developed—the redness deepening and paling with the pulse. Apart from aortic incompetence, this sign may be elicited in some degree of development under conditions of the pulse that resemble those characteristic of the lesion, for instance alcoholic muscle failure of the heart.

In examining the chest of a patient suspected of having heart disease the cardiac region, as a whole, must be carefully scrutinised. In young subjects of advanced heart disease the whole region is often prominent, owing to the enlarged organ opposing the effects of atmospheric pressure upon the framework of the thorax. This is especially noticeable when lung-expansion has been interfered with, and pigeon-breast has resulted. Again, the whole cardiac surface may seem to be thrust forward *en masse* during the cardiac systole, in which case the special visible areas of ordinary cardiac pulsation, to be described, cannot be distinguished, the condition being the result of a growth encroaching on the posterior mediastinum and pushing the heart forwards. It must be remembered that during diastole the heart is flaccid, while during systole it assumes a special shape, to accommodate which the parietes have to give way before the hardened mass of muscle the ventricles in systole represent. Even when a large aneurysm in the descending thoracic aorta projects the heart forwards against the chest wall, it is the pulsation of the heart itself of which we have chief evidence, though the two pulsations—that of the aneurysm and that of the heart—may be so intimately blended as to be indistinguishable. Apart from such rare conditions it will be found that the visible impulses of the heart are usually

three in number, and rarely there is a fourth: (1) that of the apex situated normally in the 5th intercostal space, well within the left mammary line, and occupying a small area about that of a square inch. The apex-beat represents the contraction of the left ventricle, and when this chamber is enlarged the apex-beat is displaced downwards and to the left, and remains well defined, unless the ventricle becomes rounded in shape from dilatation, in which case the apex-beat loses its definition and often disappears altogether. Some individuals, though apparently free from heart disease, have no apex-beat. This has been explained in various ways, but the fact must be recognised. In effusion into the pericardium the apex-beat may be elevated, but it is difficult in such a case to be sure that the impulse taken for the apex-beat really represents it. (2) An ill-defined pulsation below the xiphoid cartilage is known as the "epigastric impulse," and indicates the movements of the right ventricle. Certain individuals, apparently free from heart disease, have marked epigastric impulse, presumably because of their having a short sternum. This impulse is present in nearly all cases of heart disease in which there is interference with the pulmonary circulation, and is apt to be specially pronounced in cases of pulmonary emphysema, in which there is not only obstruction in the lung-circulation and engorged liver, but a lowering in the position of the diaphragm as well. This impulse of the right ventricle must be carefully distinguished from actual expansile pulsation of the liver itself, which is an extreme result of obstruction in the pulmonary circulation, and which has been already referred to. (3) The third area of cardiac pulsation to be noticed is in the 2nd left intercostal space close to the sternum. Pulsation here is probably never normal, though it may be the left lung that is at fault and not the heart. This pulsation is usually associated with epigastric pulsation, and depends on enlargement of the infundibulum of the right ventricle. At one time such pulsation was attributed to the left auricle, the appendix of which comes to the front in this situation to the left of the pulmonary artery. But the appendix is often found plugged with clot, and even when active and dilated, gets as it were pushed aside by the dilated infundibulum. Paradoxical though it may seem, as good a case for the *right* auricular appendix being the seat of pulsation in the 2nd space to the left of the sternum may be made out, for it has actually been found greatly dilated and free from clot to the left of the sternum. Those who advocate the auricular origin of visible pulsation in the 2nd left interspace, admit that such pulsation is ventricular-systolic in rhythm, and account for it by a backward current into the left auricle from the left ventricle through the mitral valves, although the systolic murmur audible over the pulsation is quite absent from the apex, to which the downward directed valve curtains might be expected to direct it. Pulsation in the 3rd left interspace is specially common, apart from mitral stenosis, in cases of chlorosis, in which the blood would seem to have difficulty in passing through the pulmonary circuit. These are just the cases in which visible venous pulsation in the neck is so manifest, bearing out the special implication of the right side of the heart in the circulatory disturbance. (4) In cases of great enlargement of the right chambers the *right auricle* occasions visible pulsation to the right of the lower half of the sternum.

Whenever the left ventricle is unduly exposed, whether it be from retraction of the left lung or from dilatation of the heart, it is common to see systolic retraction of the intercostal spaces *above* the apex-beat, a result simply of atmospheric pressure, the heart, of course, occupying less space during its systole than during its diastole. This must not be confused with retraction of the area corresponding to the left ventricle *including the apex*, which is a sign of complete pericardial adhesion, internal and external.

Palpation.—The apex-beat of the heart is the first object of attention when the hand is placed over the cardiac region. Valuable information is afforded in this way as to the condition of the most important chamber of the heart—the left ventricle—for the apex of the heart is formed alone by this chamber. The *situation*, the *extent*, and *force* of the apex-beat must in all cases when possible be carefully investigated,—that is to say, if the apex-beat is present, because in not a few cases of cardiac disease there is no apex-beat, and the negative fact is always to be noted, though it need not be given an undue share of attention, seeing that not a few individuals, who present no evidence of cardiac disease, do not possess an apex-beat. When there is an apex-beat its situation fixes at once the greatest extension of the left ventricle to the left, for the apex must always be the portion of the chamber lying to the extreme left. The extent of the impulse, which normally should occupy the space of about a square inch, again, is often increased considerably, as is specially noticeable in cases of aortic incompetence in which there is dilatation and hypertrophy of the left ventricle. A well-defined though extensive apex-beat may be taken as an indication that the ventricle still retains its form and has not become rounded or globular.

For reasons already referred to, absence of the apex-beat cannot be accepted implicitly as a sign of cardiac weakness, but if a previously present apex-beat ceases, it may be accepted as such a sign, and in corroboration of this inference it will commonly be noticed that the impulse of the right ventricle increases as that of the left declines, as was long ago observed by Stokes in typhus cases.

When the auscultatory sign, to be described later as the *bruit de galop*, is present, it is not very rare to feel over the apex a double shock during the cardiac diastole; if palpation precedes auscultation, it may be predicted in a case of the kind that the *bruit de galop* will be heard (*vide* "On a Rare Combination of Physical Signs," *Practitioner*, September 1896).

As a very rare occurrence in cases in which the pericardium is, internally and externally, universally adherent, there is no apex-beat proper, but in place of it there is during systole a general depression, *including* the region of the cardiac apex, followed by an impulse during diastole—apparently the result of the rebound of the relaxing and expanding heart. It is all-important to remember that there is no apex-beat present in the case referred to, so that this rare condition may not be confounded with the common inspection-sign of systolic recession *above* the apex-beat, that means only exposure of the heart, whether from cardiac enlargement or lung retraction.

To the observer with *tactus eruditus*, examination of the apex-beat by the hand is fraught with useful information in most cardiac cases. Apart from any accompanying thrill (to be considered later) its sharp, short stroke in mitral stenosis is most suggestive of this lesion, while in the hypertrophied heart of chronic Bright's disease the displaced, deliberate, slow heave is no less so.

Epigastric Impulse.—The impulse of the *right* ventricle is of an altogether different kind from that of the left ventricle, which is the apex-beat. It is a diffuse impulse, felt below the xiphoid cartilage, and appeals in most cases to inspection rather than palpation. Only in cases of great hypertrophy of the right ventricle does it become in any sense strong and "heaving," and in the latter case it is easily perceptible over the lower part of the sternum itself. In cases in which the right chambers of the heart are greatly hypertrophied and dilated, moreover, there is not infrequently expansile pulsation of the liver, so that there is apt to be much confusion of the latter pulsation with that of the right ventricle itself. The very rhythm of the impulse of the right ventricle has been and is still in dispute—some contending that it is diastolic and due to the inrush of blood. It is to be noted in this relation that the impulses of the two ventricles are seldom both pronounced at the same time. Again, in cases of great enlargement and hypertrophy of the left ventricle, with little or no implication of the right chambers, an abnormal impulse of the left ventricle may be perceptible in the epigastrium, and is then very liable to be taken for the impulse of the right ventricle. This happens specially in cases of aortic incompetence.

The impulse of the infundibulum of the right ventricle and that of the right auricle come under consideration as inspection- rather than palpation-signs, though they may be perceptible by means of the latter method. They have already been considered under inspection.

The shock resulting from the closure of the pulmonary semilunar valves is often perceptible on palpation in the appropriate area—the sternal end of third left cartilage—situated directly over the valves which lie very superficially. In cases of dilated aorta a similar impulse may be perceptible in the aortic area, which, however, be it remembered, is not situated over the valves, but at a distance from them, so that the perception of such an aortic impulse is much less common.

Thrills which are perceptible by means of palpation are usually represented in auscultation by corresponding murmurs, with which in rhythm and other respects they correspond. Thus in cases of mitral stenosis there may be presystolic and diastolic thrills at the apex, which can sometimes more easily be separated by palpation than the corresponding murmurs by auscultation: the *crescendo* character and abrupt termination with the apex-beat of the presystolic, and the *diminuendo* character of the diastolic murmur, are easily recognisable by both methods, but a pause between the two is sometimes perceptible by palpation, that is absent in the case of auscultation. Evidently the later vibrations of the diastolic murmur in this case are imperceptible in palpation, as one can readily understand them to be. Diastolic thrill may be present at the apex in cases of free aortic regurgitation, when the history of the case and the usual vascular indications of the lesion will commonly save the observer from error with regard to the origin of the thrill. In excessively rare cases of simple cardiac dilatation, without aortic incompetence or mitral stenosis, a diastolic thrill has been felt, as a diastolic murmur has been heard (*vide Practitioner*, vol. lii. p. 254, 1894).

Systolic thrill in the *aortic region* is common in cases of aortic stenosis. In the aortic region the systolic murmur that is commonly associated with dilatation of the arch may be accompanied by a similar thrill which the abnormal approximation of the vessel to the surface makes easily perceptible. Occasionally a systolic thrill is present at the *apex* in cases of mitral stenosis accompanied by corresponding systolic murmur, which is usually at the time the only murmur present. Possibly, an apex systolic thrill may be felt in any case of mitral regurgitation with very loud murmur.

Percussion.—In the course of a systematic physical examination of the heart, it will often happen that inspection and palpation have furnished abundant evidence of the organ being enlarged before percussion is reached. But in not a few cases the estimation of the size of the heart must be essentially based on the percussion

result alone, neither visible nor palpable impulse being produced, owing to the depressed vigour of the heart muscle and the alteration in the shape of the left ventricle, that is so apt to be associated with habitually incomplete systoles. Consideration of the heart *in situ* makes it at once evident on what principles the physical method of examination by percussion is founded. Except inferiorly the heart is surrounded by the resonant lungs, the anterior borders of which embrace the organ in such a way that a large portion of its anterior surface is covered by them. Moreover, these covering borders of the lungs are wedge-shaped, as they interpose themselves between the heart and the chest wall, that is to say, they become progressively thinner towards their edges. The anterior margins of the lungs separate at the level of the fourth cartilage, the border of the left lung passing outwards and downwards to a point situated over or close to the junction of the fifth rib with its cartilage. The border of the left lung then proceeds downwards and inwards along the sixth cartilage. Thus a more or less triangular space is formed in which the heart comes into direct contact with the chest wall, lung no longer intervening. The lower boundary of the heart, of course, corresponds to the upper boundary of the liver, the two non-resonant organs being indistinguishable by percussion. If, however, we define the upper boundary of the liver dulness to the right of the sternum, taking care to get beyond the right border of the heart—the right auricle—and from the level of the hepatic upper boundary draw a line, slightly sloping downwards, across the chest to the left anterior axillary line, such line will give an approximate representation of the lower boundary of the heart, a fact that is often readily demonstrated by the apex-beat being found immediately above it.

There are three directions in which it is necessary to determine the boundaries of the heart by percussion in order to estimate its size and ascertain its shape for practical purposes. From the size and shape of the dull area we infer the relative condition of the cavities of the heart. It is, of course, evident that the accumulation of fluid in the pericardium or pleuræ would render the estimation of the size of the heart by percussion impossible for the time being. The position of the right border of the heart should be ascertained above the upper boundary of hepatic dulness by percussing —say an inch above this boundary—from the right mammary line towards the sternum, and noting the first decided diminution of volume in the lung resonance that occurs as the sternum is approached. It is only when the right auricle has become so hugely distended as to be uncovered by lung to the right of the sternum that actual *dulness* is obtained on percussion of this region. Anatomically we know that the right auricle in health passes about half an inch to the right of the sternum, thus fully an inch from the median line, but it is covered by such a thick layer of lung here that its presence fails to modify the pulmonary percussion sound. When, on the other hand, we find absolute dulness to the right of the sternum, we are entitled to conclude that the right auricle is greatly distended and probably paralysed, while the degree of its distension is an excellent indication of the amount of obstruction in the pulmonary circulation. When there is no absolute dulness to the right of the sternum, but only a diminution in fulness or a certain "emptiness" of the resonance, as is indeed the common condition in all but most grave cases of heart disease, it is more difficult to fix the exact spot at which the presence of the auricle first modifies the pulmonary resonance, and this difficulty is increased by the approximation of the cartilages as they join the sternum, especially in certain individuals, exerting an influence on the percussion sound. Nevertheless by careful percussion towards the middle line, above the hepatic dulness, the point to which the right border of the heart extends can usually be ascertained with a fair amount of precision. Percussion up to the middle line, that is to say, over the sternum, is unsatisfactory, inasmuch as this bone has a peculiar (so-called osteal) resonance of its own, which interferes with the determination of the percussion sound that would otherwise be yielded by the underlying structures.

The next determination to be made is the greatest extent of cardiac dulness to the left of the sternum. A moment's consideration will make it plain that the portion of the heart that extends farthest to the left of the sternum must be the most important chamber of all—the left ventricle—and that its extremity represents the apex of the whole organ. Moreover, the greatest extension of the heart to the left will necessarily, owing to the shape of the organ, be at its lowest part in the vertical line of the body. When, then, percussion is made just above the level of the inferior border of the heart and from the left anterior axillary line towards the sternum, the first part of the heart that will modify the pulmonary percussion sound will necessarily be the apex, formed by the left ventricle alone; and, therefore, the greatest extension of the cardiac dulness to the left will represent the size of the left ventricle. When the apex-beat is discernible, percussion can be made directly towards it from the left anterior axillary line. Where there is no apex-beat the position of the apex must be approximately determined, as above described, by drawing a line across the front of the chest, from immediately above the upper limit of hepatic dulness to the left axillary line, almost horizontally, but sloping very slightly downwards. The first dulness met with in per-

cussing from the left axilla towards the middle line just above this line represents the apex of the heart, and therefore of the left ventricle.

There remains to be determined the summit of the cardiac area of dulness. This determination should be made in the so-called parasternal line—a line drawn vertically downwards an inch to the left of the sternum. Extension of the cardiac dulness upwards in this situation depends commonly on effusion into the pericardium (a condition not considered in this article), or on enlargement of the infundibulum of the right ventricle—conus arteriosus. Normally there should be no extension of the cardiac dulness above the third cartilage. Of course, all these remarks presuppose air-containing lung parenchyma in the neighbourhood of the heart.

In noting the features of a cardiac case, the size and shape of the heart form most important points in the formation of the diagnosis. For all practical purposes measurements in the two directions indicated above, and the noting of the rib to which the cardiac dulness reaches upwards, afford a perfectly sufficient statement of the size and shape of the heart. The results can be noted in a very brief yet perfectly satisfactory manner thus: The costal cartilage to which the cardiac dulness reaches upwards in the left parasternal line is stated in Roman figures III. or II., as the case may be, and a line is drawn underneath, as under the numerator of a fraction. Underneath this line is placed, in Arabic figures, and to the observer's left, the distance stated in inches to which the right border of dulness extends to the right of the middle line, and to the observer's right the distance, stated in inches, to which the dulness extends to the left of the middle line at the level of the cardiac apex, which is of course the extremity of both the left ventricle and the whole heart. The note is made as if it were sketched in chalk on the patient's chest.

In the normal heart there is practically no dulness to be detected to the right of the sternum. Moreover, the difficulty of detecting a difference, in their percussion sound, between the right and left halves of the sternum, as it lies over the heart, is great and often insuperable. It suffices, then, to note any impairment of resonance there may be to the right of this bone, and to measure the extent of such impaired resonance in inches from the middle line at the level indicated. When any absolute dulness is found beyond the right sternal border it may be assumed at once that the right auricle is much enlarged. The writer has known such dulness to extend almost to the right mammary line, the right auricle being found post-mortem to have become a huge thin-walled sac, in all probability incapable of systole. Absolute dulness, of course, signifies that the distended chamber has displaced the right lung which normally overlies it. In ordinary heart cases the cardiac dulness to the right of the middle line varies from an inch to an inch and a half. It may be assumed that increase of dulness in the direction indicated implies impaired contractility on the part of the right auricle. To the left of the sternum, four inches from the middle line may be considered the maximum measurement of a normal heart, and the dulness will reach as far only exceptionally and in very large individuals. Usually in individuals with sound hearts the maximum measurement to the left of the middle line does not exceed $3\frac{1}{2}$ inches.

Some examples of measurements in actual cases may be given in illustration of these considerations. The measurement $\frac{\text{II.}}{4—5}$, in a case of mitral stenosis, denoted great enlargement of the right auricle, which had no doubt become little more than a simple reservoir, moderate enlargement of the left ventricle, and enlargement of the infundibulum of the right ventricle. The disproportionate enlargement of the right side of the heart is well shown by the figure above, and by that to the observer's left. The measurement $\frac{\text{III.}}{3—7}$ represented the cardiac dulness in a case of aortic incompetence late in the course of the disease, the right auricle having become much distended while the left ventricle was huge. The measurement $\frac{\text{III.}}{1\frac{1}{2}–4\frac{4}{5}}$ may be taken as representing the average size of the heart in cases of mitral stenosis, when admitted to hospital with engorgement of the right auricle. The distension of this chamber, in cases of moderate severity, rapidly subsides under rest and treatment, and the corresponding measurement of dulness to the right as rapidly diminishes.

Auscultation.—The evidence afforded by this method of physical examination is too often accorded a value greater than it merits, although it must be granted that in some cases it adds a precision to diagnosis unattainable by the other methods, and in all cases it gives the finishing touches to the diagnosis as far as that is based on physical signs. At the outset of this subject the student must be impressed with the necessity of studying the physiological sounds of the heart and such modifications of them as there may be, before he attends to the adventitious sounds present. *He cannot too soon realise the fact that many heart cases run their course from first to last without the development of any murmur.* Among the variations that may be met with in the heart-sounds of apparently heart-healthy individuals there are many minor and, it is believed, unimportant ones. When the left ventricle is hypertrophied and thick-walled, the arterial tension being high, the first sound is often dull and toneless, while when the walls are thin and the cavity dilated, the arterial tension being low, the first sound is

often loud and short so as to resemble the physiological second sound. It is similar shortening of the first sound that gives the heart-sounds in fever the resemblance to the heart-sounds of the fœtus *in utero*, that was so well described by Stokes. In the gravest fevers, however, as in typhus, a further change is produced as the result of profound failure of the heart muscle, namely, disappearance of the first sound altogether. As an event of the greatest rarity the second sound may likewise cease, so that the patient would be reasonably thought to be dead but for the faint flicker of the radial or other pulse. When the ventricular contraction is abrupt and the arterial tension low, as in palpitation of nervous origin, the first sound, again, is usually loud and short. Sir William Broadbent has called attention to a condition of the heart-sounds which he regards as of peculiarly bad omen, and in which the first sound is short and immediately followed by the second sound. The first sound may be reduplicated in all degrees from that in which the double sound seems to give only a prolonged and somewhat blurred character to the first sound, to that in which there are clearly two first sounds separated from one another. *Turrup-dup* may be taken as representing phonetically such a reduplication as the latter along with the following second sound. Naturally, in all these cases it is over the ventricles that we expect the modifications mentioned to be best heard. In mitral stenosis the first sound is apt to undergo a remarkable change, which with quiet action of the heart is most significant of the lesion. Such change is commonly termed *accentuation*. The first sound becomes peculiarly loud, short, and sharp. When the presystolic murmur is present it is invariably accompanied by a first sound of this kind into which it runs, so that the two together form a combination of murmur and sound, accurately represented by the syllable "trupp" with an accentuated end, to which reference will again be made. The resemblance between a reduplicate first sound and the combination of murmur and sound referred to, may be close when the heart is acting quietly, but exciting the heart's action by exercise or otherwise usually at once brings out the accentuation of the first sound, if it does not also develop the murmur, and doubt is dissipated in the case of stenosis.

Of Murmurs.—The foregoing remarks suffice to show how much valuable information concerning the heart, both for diagnosis and prognosis, may be obtained from modifications of the physiological heart-sounds quite apart from the production of *murmurs*, as we term the adventitious sounds, that in no way can properly be regarded as physiological sounds however modified. For the most part murmurs may be described as "blowing" sounds. It is difficult to apply such a description, however, to the auricular systolic murmur, which is further *sui generis*, inasmuch as it is *crescendo* in character and runs into the accentuated first sound, which at once brings it to a close. Some murmurs assume musical quality, transiently or permanently, and others acquire a loudness that renders them audible not merely over the greater part of the patient's trunk, but actually at a varying distance from it. The quality and loudness of murmurs have not, however, been sufficiently utilised, either for diagnosis or prognosis, to render a discussion of them desirable here. Moreover, the caprice occasionally displayed by murmurs, in regard to the attributes under consideration, makes the probability small of their ever becoming of much practical importance. In the case of every murmur heard over the heart it is necessary for the observer to note—

(i.) The rhythm auricular or ventricular systolic or ventricular diastolic—in accordance with the physiological act taking place in the chamber of the heart concerned. When either of the physiological sounds remains audible with a murmur it is of great value in the determination of the rhythm of the latter.

(ii.) *The exact spot at which the murmur is heard loudest*, or, as it is called, *position of maximum intensity.* It must be remembered, however, that this does not necessarily correspond to any of the four areas that are usually associated with the four orifices and their valves.

(iii.) *The direction or the conduction of the murmur over the surface of the chest.*

(i.) Rhythm.—At the base of the heart murmurs may assume two rhythms, ventricular systolic and ventricular diastolic; and at the apex and sometimes over the ventricles they may assume three rhythms—auricular systolic, ventricular systolic, and ventricular diastolic.

The basic or arterial murmurs being the simpler will be considered first of all. During the systole of the left ventricle blood is rushing through the aortic orifice, and if a murmur becomes audible during this time we may presume that the current of blood is in some way interfered with. The term "obstruction" is often used to denote such interference, but it is open to very great objection, seeing that the aortic orifice may even be larger than normal. For instance, in aortic dilatation a very loud systolic murmur is often audible in the aortic area, of which the explanation is that, large as is the orifice of the vessel, its channel is larger still, so that the current entering through the former has to spread out to occupy the channel beyond. This condition we know to be the cause of arterial murmurs in general, so that in health all we have to do in order to produce a murmur with the blood current is to narrow the channel of an artery by pressure with the stethoscope. With regard to the aortic orifice

a systolic murmur is only too likely to be produced by a trivial lesion, which accomplishes as regards results on the circulation an altogether unimportant effect on the blood-stream—it may be a tiny fibrous nodule or thickening of the lip of a cusp, it may be mere thickening and stiffening of a cusp that prevents its natural effacement before the blood current, and, possibly, it may be a projecting atheromatous patch in the channel of the vessel beyond the valves. On the other hand, there may be extreme narrowing of the orifice, yet such grave lesion may be revealed on auscultation by a murmur of the same rhythm as, and otherwise indistinguishable from the murmur produced at an actually enlarged orifice and at an orifice only nominally diminished. (We have been constrained to use the expression "at," but "beyond" would be more strictly correct.) Lastly, as if to shatter hopelessly the value of a systolic murmur as a sign of aortic disease, not to speak of obstruction, this same systolic murmur is often heard over the aorta when it is perfectly healthy, as far as we know, in cases of anæmia. Notwithstanding, under certain circumstances having relation to accompanying phenomena, the diagnosis of aortic stenosis may to a considerable extent be based on this murmur, but even when this is the case, it must be admitted that the murmur then only contributes to the basis of the diagnosis: it does not essentially constitute it.

As a matter of clinical experience a systolic murmur in the "pulmonary area"—over or just beyond the pulmonary semilunar valves—is common, and, as described in another place, the mode of origin of such a murmur has been hotly debated. This murmur has become in clinical medicine specially associated with anæmia, in which condition of ill-health the murmur is very common. Still the fact remains that cases of anæmia in a pronounced degree are from time to time met with in which there is no murmur in the pulmonary or other area. In all kinds of heart disease, again, a "pulmonary" systolic murmur is common, and in certain cases of muscle failure of the heart it may be the only murmur audible throughout the illness, as for instance in alcoholic muscle failure.

In long-standing cases of mitral stenosis and like conditions it is not very rare for the pulmonary artery to become atheromatous and to lose its elasticity, so that dilatation is very apt to ensue. Without atheromatous change, indeed, it is likely that the artery will become dilated after prolonged exposure to high blood-pressure, and this event may, as in the case of the aorta, give rise to systolic murmur.

Even in ordinary cases of anæmia it is conceivable that this notorious systolic murmur in the pulmonary area may be produced in this way, the condition of anæmia seemingly leading to a difficulty in the passage of the blood through the capillaries of the lungs. We know how asphyxia leads to engorgement of the right side of the heart, and it is obvious that interference with the entrance of oxygen into the air-passages, and a diminution in the number or capacity of the oxygen-carriers of the blood, must both occasion some degree of interference with the pulmonary circulation and raise the blood-pressure in the pulmonary artery. Clinicists have long recognised that a systolic murmur over the pulmonary artery is the least valuable cardiac murmur with which they are familiar. It commonly accompanies other murmurs—both those dependent on valve lesions and those dependent on simple muscle failure. In a first attack of rheumatic fever it is very commonly encountered in association with a mitral regurgitation murmur, and (a fact of some interest in relation to the theory above referred to) it is commonly also associated with a tricuspid murmur. In the alcoholic heart and other forms of muscle failure, again, it may be the only murmur present. Sometimes it is present although there is no indication of ill-health of any kind, when it may be conjectured to result from some peculiarity in the contour of the vessel—some odd bend or the like. The low value of this murmur in diagnosis is firmly established, whatever may be our ideas as to the mode of its production.

Incompetence of the aortic valves is revealed to us on auscultation by a very definite *diastolic* murmur well conducted, downwards and to the left, over the cardiac area. When any portion of the second sound is audible the murmur runs off directly from it; that is to say, there is never the intervention of a pause between the second sound and the murmur.

When the blood tension in the pulmonary artery is habitually very high, as in cases, for instance, of mitral stenosis and pulmonary emphysema, the vessel is apt to undergo dilatation whether there be atheroma or not, and, as in the case of the aorta, the orifice, as well as the channel of the vessel, may finally take part in the dilatation, so that the valves fail to completely close the orifice, temporarily or permanently. Thus a murmur of pulmonary incompetence may arise as a curiosity of clinical observation—the second sound from which it proceeds being always much accentuated. This murmur of high pressure in the pulmonary artery is only an example of the same process that occasions the much more common murmur occurring under similar circumstances in the aorta.

As regards their rhythm, *mitral* and *tricuspid* murmurs may be: (1) *Ventricular systolic;* (2) *Ventricular diastolic;* and (3) *Auricular systolic*, commonly called *presystolic*.

(1) It will be convenient to consider the ventricular systolic murmur first of all. This murmur indicates incompetence of the mitral or tricuspid valves according to its localisation and position

of maximum intensity. Incompetence of the tricuspid valves is a very common consequence of disease, of one kind or another, affecting the left chambers of the heart, whereby the blood-pressure in the pulmonary circuit is much raised. In comparatively a very few cases there is stenosis of both mitral and tricuspid orifices, but the left lesion is always far in advance of the right. In such cases it is common for the simple murmurs of tricuspid and mitral regurgitation to have been the only murmurs observed during life.

In the immense majority of cases of heart disease in which the murmur of tricuspid incompetence is found, there is no lesion of the tricuspid valves at all, and their incompetence is simply the result of muscle failure of the right ventricle.

When a systolic murmur is heard at the *apex* we assume that the mitral valves have become incompetent, though the possibility of the murmur of aortic stenosis being conducted to the apex is difficult to deny altogether. Taken practically, the question is not one of great importance, and, given aortic stenosis, sooner or later the mitral valves are likely to become incompetent. In all forms of muscle failure of the heart mitral incompetence is very apt to arise, and with it the murmur we are considering. Then there are the cases in which this murmur results from actual damage to the valve curtains, rendering them incompetent. Rheumatism and septic endocarditis are the usual causes of the damage, but the latter is by far the more destructive to the valve structures, though the disease is comparatively rare; while in rheumatism, which is common, the damage to the valve is apt to be much less in the first instance, although the chronic process set up very frequently eventuates in stenosis of the orifice. Lastly, when mitral stenosis is thoroughly established, the necessarily deformed curtains, very commonly indeed, are, either permanently or from time to time, incompetent, so that the murmur of mitral incompetence is the most common of all the murmurs met with in cases of stenosis. Thus it happens that when a systolic murmur is loudest at the apex of the heart we associate it at once with mitral incompetence, and we have to ask ourselves practically these three questions: (*a*) Are the valves healthy, and is their incompetence the result of muscle failure of the heart? (*b*) Is there structural damage of the valve curtains rendering them incompetent, but without stenosis of the orifice? (*c*) Is there stenosis of the orifice?

(2) The next murmur, the rhythm of which we have to consider in relation to the mitral and tricuspid orifices, is the *diastolic*. During the diastole of the ventricles blood is passing through the auriculo-ventricular orifices, and the condition of these orifices that produces a diastolic murmur is stenosis. (This statement makes no account of the very rare occurrence of a diastolic murmur in simple cardiac dilatation alluded to in another part of this article, p. 135; *vide Practitioner*, vol. lii. p. 254.) Tricuspid stenosis is a rare lesion, and a diastolic murmur is only very exceptionally produced by it, so that we may direct our attention exclusively to the mitral diastolic murmur, which is a common sign in cases of mitral stenosis. The murmur is usually best heard at the apex, but is less strictly limited to this spot than is the murmur to be next considered, being often audible over the ventricles between the apex and the lower part of the sternum. In rare instances it is widely distributed over the cardiac surface, so that when, as is usually the case, there is an accompanying systolic murmur, the double murmur may be, as far as sound goes, indistinguishable from the familiar "double aortic" murmur of aortic incompetence (*vide Med. Chron.* (1896), vol. vi. p. 174). The diastolic murmur of mitral stenosis, unlike the murmur next to be described, is somewhat *diminuendo* in character. The current of blood through the constricted orifice producing this murmur has both a *vis a fronte* and a *vis a tergo* in its production: the expanding left ventricle and the blood-pressure in the pulmonary circuit derived from the contraction of the right ventricle.

(3) The last murmur we have to consider is the *auricular systolic or presystolic murmur* of auriculo-ventricular stenosis. Like the last murmur, it is much more frequently produced in the left side of the heart, though a tricuspid presystolic murmur does very rarely occur. In several respects, as well as in its rhythm, this murmur differs from all the other murmurs with which clinical experience has made us familiar. As regards its rhythm, it immediately precedes the first sound, thus occurring during the latter portion of ventricular diastole. It is *crescendo* in character, and it is brought abruptly to a close with the first sound (modified as we have found) *when the murmur is at its greatest intensity.* This combination of *crescendo* murmur and first sound lends itself well to the usually accepted view as to the mode of production of the murmur by the current of blood resulting from the contraction of the auricle, for we can imagine the auricle expelling the blood with increasing force as its capacity diminishes, and we can understand the abrupt cessation of murmur at the moment of its greatest loudness, for how can the thin-walled auricle contend for an instant against the mass of muscle composing the left ventricle? Evidently, directly the ventricle enters into contraction, the current of blood issuing from the constricted orifice must be brought to a standstill.

(ii.) *Situation of Maximum Intensity of*

Murmur.—In our endeavour to determine the orifice at which a murmur is produced, the place at which it is heard loudest must always be ascertained.

As a matter of fact all the orifices of the heart lie close together. To use Dr. Walshe's statement, "A superficial area of half an inch will include a portion of all four sets of valves *in situ*; an area of about a quarter of an inch, a portion of all except the tricuspid." It is therefore impossible to assign a murmur to a certain orifice because it is heard loudest over that particular orifice, seeing that the other orifices are in such close proximity, but the less important orifices—those of the right side —lie superficially, and one of these—the pulmonary—is so close to the surface that the shock of the closure of its valves can often be felt, as we have already found, on palpation. The areas at which pulmonary and tricuspid murmurs are usually best heard are, accordingly and respectively, "the sternal extremity of the 3rd left cartilage" and "the lower part of the sternum and adjoining area to the left of the bone where the heart is uncovered by lung." We expect murmurs generated at these orifices to be heard loudest in the areas named, which practically overlie the orifices concerned. As will be explained, however, under "Conduction of Murmurs," certain objections may be raised to the selection of the areas in question. It is impossible, however, to find better ones.

The principle on which the areas assigned in auscultation to the more important aortic and mitral orifices are selected is quite different, and is that of the *isolation* of the murmurs at some distance from the orifices at which they are produced. The "aortic area" is the "sternal extremity of the 2nd right costal cartilage"; here the aorta comes close to the surface, but of the two murmurs that may be generated at its orifice the systolic has unquestionably a better chance of being conducted hither in full intensity than the diastolic murmur. Clinical experience, as well as theory, bears out this statement, for one is often surprised at hearing so little in the aortic area of an aortic diastolic murmur that is loud below. The "mitral area," again, corresponds to the apex of the heart as well as the apex of the left ventricle. At the cardiac apex, then, we have to do with this chamber only. In the case of the direct or obstructive mitral murmurs, we can well understand their free conduction in the direction of the current that produces them, but for the murmurs of incompetence the apex as the place of their isolation would, at first, seem ill-chosen. It is known, however, that the disposition of the valve curtains, as lips projecting in the opposite direction to the regurgitant current, is calculated to reflect the murmur of incompetence forwards to the apex (*vide* Fagge: Reynolds' *System of Medicine*, vol. iv. p. 630). The frequent presence of this same murmur of mitral incompetence at the back is, of course, readily explained by the posterior position of the left auricle into which the regurgitated stream must directly pass.

While it is desirable that special attention should always be given both to the cardiac sounds and to any murmurs that may be audible in these four areas, auscultatory examination of the heart must not be *limited* to them. The necessity for this injunction will be evident on consideration of the following paragraphs.

(iii.) *The Conduction of Murmurs.*—The most important murmurs—the aortic and the mitral —have fairly definite lines of conduction, and especially is this the case with respect to the regurgitation murmurs of both aortic and mitral origin. As regards the obstruction murmurs produced at these orifices, the aortic is carried on in the course of the circulation, as might have been anticipated, and may be audible over the upper back in the left vertebral groove; the mitral is likewise carried onwards in the course of the circulation to the cardiac apex, beyond which it only exceptionally spreads. The systolic murmur of mitral incompetence, especially when there is structural change of the valve curtains without stenosis or very free regurgitation from muscle failure, is apt to be very well conducted to the back. On the other hand, in cases of regurgitation through a stenosed orifice, the murmur is often not conducted to the back, only a first sound being audible there, and the same condition is the rule in cases of mitral incompetence from muscle failure, the exception to the rule being found in cases in which the incompetence apparently is unusually great for the time being. Murmurs that are inaudible at the back may be audible in the axilla as far as the mid-axillary line, or even beyond it, while those audible at the back are usually heard continuously, as the stethoscope is passed backwards, from the apex to the spine. In a considerable number of cases the murmur is not only audible, but loud at the back to the right of the spine. As in these cases there is often tricuspid regurgitation as well as mitral, some have supposed that the loud murmur to the right of the spine is tricuspid in origin, but this is very doubtful.

The murmur of aortic incompetence is conducted downwards and to the left, towards the apex. In many cases it is heard over all the cardiac area, in most cases it passes well down the sternum, and it may even be most distinct at, or actually limited to, the lower part of this bone. Feeble murmurs are not rarely located to the left of the sternum between the 3rd and 5th cartilages. It will be evident that these last two positions properly belong to tricuspid and pulmonary murmurs respectively, but as regards the former a tricuspid diastolic murmur is exceedingly rare, much more rare than the

rare lesion it indicates—tricuspid stenosis—while pulmonary incompetence as an actual lesion is still more rare, so that these murmurs are little likely to occasion error in diagnosis. Moreover, in aortic incompetence, the vascular phenomena, referred to elsewhere, are generally characteristic of this lesion.

There are certain *noisy* murmurs of which conduction in definite directions can hardly be recognised, and which may indeed be audible at a distance from the patient's body. Aortic incompetence is specially likely to furnish examples of such murmurs, which may be heard all over the trunk and along the spine from occiput to sacrum, as well as at a distance from the body. How little variation as regards murmur can be attributed to changes in the lesion was well illustrated by a case of the writer's. A patient was admitted to the wards so intensely cyanotic that he was at once bled. On his entering the ward most careful auscultation failed to reveal any murmur, yet a day or two later the patient presented a systolic murmur of extraordinary loudness audible all over his chest, and even at a short distance from it. The patient had been the subject of acute rheumatism many years before, and on post-mortem examination, a considerable time after his admission to hospital, was found to have a greatly constricted mitral orifice without any trace of recent lesion. Similar caprice in the behaviour of a murmur is often witnessed in cases of aortic incompetence, in which a long-familiar murmur may disappear during febrile or other disturbance of the circulation, while it cannot for a moment be doubted, from the arterial condition, that regurgitation is taking place as freely as ever. Such cases supply a warning not to base a diagnosis of such and such condition of lesion on any peculiarity of its murmur as regards loudness, distribution, or quality. A curious point with regard to musical *quality* in the murmur of mitral incompetence is the frequent loss of this quality by the murmur audible at the back. If we draw no special inference from musical quality in a murmur this curious experience will not disturb us, but it does raise a doubt in one's mind as to the musical murmur at the apex and the non-musical murmur at the back being, as is usually believed, one and the same.

Having considered the facts to be ascertained by the physical method of diagnosis in the abstract, it will now be necessary to reconsider them as they occur in nature, grouped together in varying combinations, according to the form of disease that gives them origin.

The Physical Signs in Combination of the Different Forms of Heart Disease

We shall in this section deal, in the first instance, with one of the simplest of the valve lesions—*obstruction at the aortic orifice or aortic stenosis.* From its very nature, this lesion, which has usually been produced by rheumatic endocarditis, is of slow formation, and the left ventricle —the most important chamber of the heart—has plenty of time to accommodate itself to the adverse circumstances the lesion imposes on it. The difficulty must be encountered first of all in systole, and, so long as complete systole on the part of the left ventricle is maintained, the difficulty is limited to systole. Only when systole fails in its perfect accomplishment because of leakage through the mitral curtains as a result of muscle failure, or because of imperfect emptying of the ventricle as a consequence of this same muscle failure, does occasion for dilatation of the left ventricle arise. The importance of these considerations in regard to the physical state of the heart in aortic stenosis is immense, and by them we can understand how it happens that this lesion of all others has been notorious for the length of time it can be endured with comparative impunity. When once, however, muscle failure has set in, the gravity of the lesion is great. The slow pulse that is frequently noticed in cases of the kind perhaps helps in warding off the development of muscle failure, and may be regarded as a manifestation of the *vis medicatrix naturæ.* Coming to a systematic investigation of the physical signs met with in a case of aortic stenosis, our first inspection should be made of the *neck*, in which arterial pulsation tends to be less evident than in most other forms of heart disease, while the presence and degree of venous pulsation depend on the development of muscle failure of the heart as a whole—in other words, on the implication of the right chambers—in the circulatory disturbance. On inspecting the chest the apex-beat may or may not be discernible; its situation in the former case will determine the size of the left ventricle, while epigastric impulse points to involvement of the right ventricle in the results of the lesion. It has been held that aortic stenosis tends to diminish the apex-beat, and in some degree this may be the case, but the vigour of the muscle of the left ventricle as well as the shape of the chamber has always to be taken into account with regard to the apex-beat. Inasmuch as aortic incompetence is often associated in some degree with stenosis, it may be well to bear in mind that incompetence would seem to tend in the direction of exaggerating the apex-impulse. *Palpation* will usually confirm the observation made by inspection as regards arterial pulsation in the neck, and the force, extent, and situation of such cardiac impulse as there may be. The characters of the pulse are reserved for special comment later. Over the aortic area a very valuable palpation sign may often be elicited, namely, systolic thrill, which may be felt all over the course of the aorta, from its origin deep in the heart to the termination of its contiguity with the upper part of the sternum

at the 2nd costal cartilage, no doubt transmitted in part of the vessel's course through the pulmonary artery and the conus arteriosus, while it may be felt on a finger, in the suprasternal notch, being thrust behind the manubrium sterni. It must be remembered that a similar systolic thrill may be met with under conditions the very opposite to those of stenosis, namely, cases of aortic dilatation, of which condition exaggeration of pulsation and the auscultatory condition to be described will usually afford ample evidence. It may be said that aortic stenosis militates against aortic dilatation, but the two conditions are not incompatible, as a matter of pathological experience. *Percussion* affords information as to the size and shape of the heart, and therefore as to the condition of its chambers in regard to dilatation. It need not be here referred to in detail.

Auscultation affords very valuable evidence as to the presence of aortic stenosis, though the murmur—systolic—associated with the lesion is of itself of little value, and for this reason, that under the opposite condition—that is to say, dilatation of the orifice—the same sign may be pronounced. Such dilatation invariably involves the first part of the arch, and therefore the second sound is intensified and accentuated, while in aortic stenosis the second sound is ill-developed, feeble, and toneless as a rule, and in many cases entirely absent, and this too without there being any diastolic murmur. It must also be remembered that diastolic murmur is not rarely associated with dilatation of the aorta and consequent accentuation of the second sound, with which character the incompetence of the valves does not usually appreciably interfere. It follows that if we meet with a case in which there is a systolic thrill and a loud, harsh systolic murmur, followed by a feeble second sound, or no second sound at all, in the aortic area, the probability of there being aortic stenosis is great. The presence of slight diastolic murmur will rather enhance than impair the value of this evidence. The history of an old rheumatic attack still further strengthens the evidence, as do also the minor physical facts elsewhere referred to, such as slow pulse, deficient carotid visibleness, etc.

The Pulse in Aortic Stenosis.—There are two varieties of pulse that have become associated

Fig. 1.—Anacrotic pulse of aortic stenosis.

in clinical medicine with aortic stenosis; probably neither possesses pathognomonic value in diagnosis, and to one of them greater diagnostic significance is to be attached than to the other. These are commonly known as the (1) anacrotic pulse and the (2) bisferiens pulse, and as regards their diagnostic value they stand in this order. The characters of these pulses are best studied graphically with the aid of the sphygmograph. (1) The sphygmogram of the anacrotic pulse is characterised by a sloping upstroke, which near the top is marked by a slight wave indicative of the position of the percussion wave, but the actual apex of the curve is above this, and is formed, not as normally by the percussion wave, but by the tidal wave, which is usually rounded and prolonged. The line of descent, like the upstroke, is sloping, and the dicrotic wave in its course is apt to be ill-marked. The pulse is usually infrequent, and has besides a sluggish wave-beat. When from nervous or other influence the pulse is rendered frequent, the characteristic features of the sphygmogram may become very much altered; on the other hand, a great degree of persistence of these same characters in spite of disturbing influences, as those of vaso-dilators and pyrexia, may be manifest.

When the anacrotic pulse is well developed and is associated with the characteristic murmur and thrill of the lesion, its diagnostic value is great. It must be remembered, however, that most pulses with a pronounced tidal wave are readily rendered anacrotic by the application of too great pressure with the instrument. The sensation imparted through the finger by an anacrotic pulse is sufficiently denoted by the sphygmogram. The infrequency and slowness of the pulse are its special features.

(2) The bisferiens pulse possesses very different features, and it certainly bears less definite relationship to the lesion than the anacrotic pulse. The upstroke is perpendicular, the apex of the percussion wave acute, and the cleft between this and the tidal wave remarkably deep, approaching in well-marked examples the "respiratory line," while the tidal wave itself is little less peculiar, its summit forming an acute angle while the aortic notch is situated low down in the line of descent of the curve. The dicrotic wave is usually, but not invariably, ill-developed.

Fig. 2.—Bisferiens pulse of aortic stenosis. Valves incompetent.

This pulse can easily be recognised by palpation simply, at least the double beat can, though it may be impossible to distinguish the nature of the second beat in this way, that is to decide whether it is the tidal or the dicrotic wave that has become exaggerated. Exaggeration of the dicrotic wave in a case of aortic disease is, however, unlikely.

Aortic stenosis is often associated with some degree of incompetence of the valves, and this may be so developed as to modify the characteristic tracing of the more pronounced lesion.

Aortic Incompetence.—Incompetence of the aortic valves differs considerably from aortic stenosis with regard to its effect on the chamber of the heart that is immediately and chiefly concerned—the left ventricle. The changes that occur in the latter are dilatation and hypertrophy. Very little consideration of the mode in which these changes are brought about will render it evident that the former process must be the first established, and there is no difficulty in accounting for the latter process, the former once established. With the advent of aortic incompetence, the ventricle has to accommodate the regurgitated blood *plus* the normal quantity, if the circulation is to be maintained. But the larger the cavity of the ventricle the greater must be, by physical law, the contractile power requisite to empty it. These theoretical considerations are abundantly borne out by clinical experience. It may be said, indeed, that aortic incompetence is the lesion of all others that is found associated with a huge left ventricle. As the muscle of this chamber begins to fail in vigour, secondary results of such failure become manifest, and take the usual forms of mitral regurgitation and imperfect systole, which in their train bring disturbance of the pulmonary circulation and engorgement, and finally dilatation of the right ventricle and auricle, usually associated with incompetence of the tricuspid valves and imperfect systole from muscle failure. Of course in a rheumatic case it is always exceedingly probable that the mitral valves are independently damaged and deformed, rheumatic endocarditis seemingly having a preference for the mitral valves.

Inspection.—One of the most notable signs of aortic incompetence is the exaggeration of arterial pulsation, which is perhaps nowhere better displayed than in the neck. The upper portion of the neck is the best to observe, in looking for this sign, as in the late stages of the disease venous pulsation may be pronounced as well, and the latter is most noticeable at the base of the neck. So long as the auricle retains its contractile activity the venous pulsation will appear to be double or undulating, as already explained, while the slightest pressure over the vein at the root of the neck at once stops the venous pulsation, the arterial pulsation remaining unaltered. The peculiar throb of the carotids at the upper part of the neck is one of the most striking clinical signs of this valve-lesion, and depends on the wide variation between the maximum and minimum blood-pressures that results from it. All the arteries of the body participate in the character, but the larger vessels are those to which attention should be directed. Very rarely various superficial veins may be seen to pulsate, but the same phenomenon is occasionally met with in other forms of cardiac disease.

Under inspection must be mentioned a minor sign of aortic incompetence already described, namely, the so-called "capillary pulsation" that becomes visible when a patch or streak of erythema is produced on the forehead by rubbing a hard substance, as the finger-nail, over the skin. The erythema is seen to redden and pale with the heart-beats.

Proceeding to the examination of the cardiac region the increased extent and displacement of the apex-beat will usually bear witness to the enlargement of the left ventricle. Epigastric impulse will be more or less pronounced according to the degree of implication of the right ventricle. In a few cases the impulse of a huge left ventricle would seem to be predominant even in the epigastrium, but in such cases the left ventricular impulse is widely diffused, while the indications of much implication of the right chambers are absent or ill-marked. Pulsation of the liver itself, again, is much less likely to be met with in aortic than in mitral cases. Our considerations led us to the conclusion that there is no lesion that more readily dilates the left ventricle than aortic incompetence, and when the ventricle is not only large, but has lost its shape and become rounded, a true apex-beat tends to disappear, and the impulse of the left ventricle that remains is diffuse and extensive. On the other hand, all visible impulse may cease, an indication of failing power on the part of the heart muscle, especially the portion forming the left ventricle. The differences with regard to the natural impulse that are met with in individuals must be borne in mind: what is referred to specially here is progressive change in the course of the case.

If the aorta becomes dilated, pulsation may become visible in the suprasternal notch, and in the second and first interspaces close to the sternum, a sign essentially due to the enlargement of the vessel, though promoted by the incompetence of the valves.

Palpation is useful chiefly to confirm the indications afforded by inspection. The force of the visible impulses can be better estimated by it, and so an inference be drawn more correctly as to the power of the heart muscle. Thrills are also ascertained by it. Over the aorta there may be a systolic or a diastolic thrill, or both. A systolic thrill—due to dilatation—is the more common in this region, though in rare cases a diastolic thrill is perceptible, and, curiously, may be limited to the apex. Such a thrill as this last probably in no way entitles to the assumption of any special condition of lesion in the valves, such as a long tag hanging down into the ventricle, etc., for the writer has found a diastolic thrill at the apex when the aortic incompetence was due to dilatation of the aorta and its orifice only, and was unassociated with any valve lesion—aortic or mitral. When the ventricular impulses, especially the left, are invisible, the *tactus eruditus* of a skilful observer

may yet afford him information as to the power and size of the chambers.

Percussion.—By this method the usual preponderating increase in the size of the left ventricle can be demonstrated, the extension of cardiac dulness taking place towards the left—to the left with an inclination downwards more or less marked. That this should be so is evident from the anatomy of the heart. In cases in which the right chambers have become secondarily enlarged, the extension of the right auricle to the right of the sternum will enable the observer to gauge the dilatation suffered by this chamber and the degree of obstruction in the pulmonary circuit that has occasioned it. When dilatation of the aortic arch has reached a high degree, a "mediastinal" dulness—that is to say, a dull area passing across the middle line in the upper sternal region, and reaching to neither "costo-acromial" angle—may be detectable.

Auscultation.—The great sign that this physical method of diagnosis affords in aortic incompetence is a diastolic murmur, heard in some part of the triangle defined as follows: by a line drawn along the right border of the sternum from the second costal cartilage to the xiphoid cartilage, and by lines joining the extremities of this one with the apex of the left ventricle, which is, of course, the point of greatest extension of the whole heart to the left. Over all this triangle, or limited to any part of it, the diastolic murmur of aortic incompetence is audible. Most murmurs of aortic incompetence are well heard down the sternum and to the left of the lower half or more of the bone. Feeble murmurs, again, may be limited to a small area close to the left sternal border, lying between the third and fifth cartilages. Some aortic murmurs are transmitted specially to the apex, and these have to be carefully distinguished from certain mitral diastolic murmurs, the resemblance being increased by the possible association of both murmurs with an accompanying thrill. In all cases of suspected aortic incompetence it is necessary to explore the whole of the triangle defined above. The majority of cases of aortic incompetence also present a systolic murmur accompanying or replacing the first sound of the heart in the aortic area. This, as already emphasised, must not be accepted as a sign of aortic stenosis, inasmuch as it may be produced by the most trivial of projections on a valve or some little thickening or rigidity of the same, in which cases the obstruction is only nominal, or the same murmur may be met with, as already explained, when the orifice, far from being smaller than natural, is actually larger than normal, in dilatation of the arch. This last type of murmur may be not only very loud, but heard over a considerable area, the aortic arch being in contact with the chest wall to an abnormal extent. (Thrill may accompany such a murmur and be felt over an extensive area corresponding to the dilated vessel.) Valuable information as to the actual condition of the affected parts may be obtained from a consideration of the second sound of the heart when that is still present, while its absence probably indicates destruction or profound deformity of the valves. The alteration of the second sound that is of chief diagnostic importance is accentuation or intensification. The combination of a systolic murmur followed by such a second sound renders dilatation of the arch, involving the ascending portion, almost certain, and the preservation of the sound in this form indicates such a degree of integrity of the valves as to render it very probable that the incompetence depends rather on dilatation of the aortic orifice than upon valve change. On theoretical grounds it has been assumed by some that incompetence according to its degree interferes with accentuation of the second sound. Clinical experience fails to support such assumption, and one thing is absolutely certain: accentuation of the aortic second sound and diastolic murmur are frequently associated, the murmur following the second sound without the intervention of the slightest pause. Dr. Walshe long ago called attention to an alteration of the aortic second sound that he conceived to be due to a short murmur preceding the second sound and running into it, as the mitral presystolic murmur runs into the first sound, and such alteration he no doubt correctly associated with dilatation of the aorta. The writer recognises the altered sound referred to, and the accuracy of Dr. Walshe's inference as to its pathological association, but he regards it rather as a modification of accentuation than as a combination of murmur with sound. Phonetically, the sign referred to is represented, according to Dr. Walshe, by the letters *phwi . . . tt.* In this relation it is curious to note that it is often difficult to distinguish simple accentuation of the first sound in mitral stenosis from the combination of short presystolic murmur running into an accentuated first sound.

The Pulse in Aortic Incompetence.—Long before the days of the sphygmograph the peculiar characters of the pulse in aortic incompetence had attracted the attention of physicians. Sir Dominic Corrigan in 1838 first described these characters in relationship to the lesion, and throughout the medical world the pulse is known as "Corrigan's pulse." Perhaps the most concise statement with regard to its characters is that they consist of a very extensive and a very rapid transition from the maximum to the minimum blood-pressure. The term "the pulse of unfilled arteries" is objectionable, seeing that the arteries are never empty; the name "Corrigan's pulse" implies no theory, and does honour to one to whom

honour is due. When the finger is laid on the pulse in aortic incompetence the stroke or "ictus" of the pulse is much exaggerated, not only by the rapidity of its onset, but by the shortness of its duration and its abrupt cessation. Thus the pulse is one moment full and strong, the next it has collapsed. These features are intensified by raising the patient's arm, which renders the collapse of the pulse more evident. If the front of the forearm is grasped by the observer's whole hand the pulse is easily felt up to the flexure of the elbow. The visibleness of the arterial pulsation has already been referred to (Inspection).

The sphygmogram possesses very different features from those it presents in cases of aortic stenosis. Only in one minor character do the sphygmograms usually agree, and that is in the tendency to ill-development of the dicrotic wave. The upstroke is perpendicular, the percussion wave exaggerated, and the line of descent precipitous. The tidal wave varies; sometimes it is rounded and sustained, while the dicrotic wave is deficient, and the line of descent slopes more than usually. This type of tracing the writer believes to point to dilatation of the aorta being the essential lesion, the incompetence being secondary in sequence as well as in importance. The other type of tidal wave is characterised by its summit forming an acute angle, as in the bisferiens pulse, although the cleft between this wave and the percussion wave is much less deep, while the tidal wave itself rises less high. It has been suggested that the two types of pulse tracing described may be diagnostic of the lesion commencing in the channel (dilatation) and in the valves of the aorta respectively, but the writer is satisfied that reliance cannot be placed on this diagnostic point.

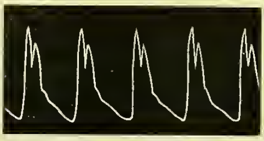


Fig. 3.—Typical pulse of aortic incompetence. Percussion wave well developed; dicrotic wave almost absent.

Again, it is necessary to bear in mind that the lesions—aortic obstruction and incompetence—are apt to be associated in all varieties of proportion, and may be so equally developed that the tracing ceases in any way to be characteristic of either lesion.

Mitral Stenosis.—The physical signs of mitral stenosis:—*Inspection* reveals little that is distinctive of the lesion, unless it be in a few cases, the depth of cyanosis, such as is reached, leaving congenital disease out of consideration, only in one other common disease, namely, pulmonary emphysema. The muscular hypertrophy of the neck, and the labouring of respiration in this latter disease, are, however, likely to be so pronounced as at once to indicate the primary lung disease. In mitral stenosis venous pulsation in the neck is usually well developed, and along with it some exaggeration of arterial pulsation is commonly present. The apex-beat is often visible and fairly well defined, and displaced towards the left rather than downwards, while epigastric pulsation (R.V.) is usually evident. Sometimes in severe cases a fulness due to the enlarged liver is easily discerned on inspection, and is seen to undergo expansile pulsation. Such pulsation must be distinguished from the ordinary form of epigastric pulsation resulting from the movements of the right ventricle enlarged and hypertrophied. Sometimes pulsation is visible above the third left cartilage, due to dilatation of the infundibulum of the right ventricle.

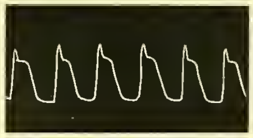


Fig. 4.—Typical pulse of aortic incompetence. Dicrotic wave absent.

Palpation will usually confirm many of the observations made during inspection: for instance, the position and definition of the apex-beat, the exaggerated impulse of the right ventricle in the epigastrium, and—it may be—true pulsation of the liver. At the cardiac apex valuable information may be obtained from the presence of *thrills*, which may be in rhythm, presystolic, diastolic, or systolic. The last is the rarest and of least value in diagnosis, and may partake of the caprice so characteristic of the auscultatory signs of mitral stenosis. The presystolic and diastolic thrills are commonly associated, and are easily distinguished with the hand, the former running up to the apex-beat with which it ceases, the latter following the apex-beat after a short interval, while it often runs into the presystolic thrill, which, however, is easily distinguished by a fresh and increasing accession of strength until it is abruptly cut short by the apex-beat. The thrills—presystolic and diastolic—described are often quite as characteristic of the lesion as the corresponding murmurs. The shock of the pulmonary semilunar valve closure is often perceptible on palpation.

Percussion will indicate approximately the size and shape of the heart. As preponderance of the right chambers is common, increase of dulness to the right of the sternum, representing dilatation of the right auricle, is the rule; the right ventricle, however, occasionally directly modifies the percussible outline of the heart in a curious way, for the change is noticed to the *left* of the sternum; in long-standing cases the conus arteriosus or infundibulum becoming greatly enlarged, so that the origin of the pulmonary artery is elevated, and the cardiac dulness passes above the third left cartilage. Enlargement, to some extent, of the left ventricle is common, as a matter of experience, in cases of mitral stenosis. Exceptionally, however, it must be admitted one finds that percussion

indicates the large right chambers and small left ventricle that theory would lead one to regard as characteristic of the lesion. When in a long-standing case of mitral stenosis seen for the first time, enlargement of the cardiac dulness in the three directions—upwards, to the right, and to the left—may cause the dull area to resemble in shape and size the dull area due to pericardial effusion, as Sir Wm. Gull long ago pointed out. The average size of the heart in fifty-five cases of mitral stenosis was $\frac{\text{III.}}{1\frac{1}{2}-4\frac{4}{5}}$.

Auscultation.—The cardiac sounds always to be given precedence, in relation with murmurs, in the auscultatory examination of the heart often furnish evidence of chief importance in cases of mitral stenosis. The first sound at the apex is apt to be abrupt, accentuated, and short, and often possesses these characters quite apart from association with the presystolic murmur, of which it must be admitted such a modified first sound forms an integral part. The second sound in the same situation—the "apex-beat"—likewise often undergoes modification, and in one or other, it may be said, of opposite directions, for it may be doubled or it may be absent. In the former case it may well be doubted if the nomenclature is correct, and if we are dealing with the physiological second sound of the heart at all. The second sound of the pulmonary artery is usually, apart from reduplication, accentuated, the closure of the valves being often so forcibly effected as to communicate a perceptible shock to the palpating hand placed over the valves which lie superficially. The reduplication of the second sound (so-called) in mitral stenosis is often best heard to the left of the lower half of the sternum, and the relationship of such a reduplicated diastolic sound to the like sound met with in combination as the *bruit de galop* in muscle failure is of great interest. It rarely happens that in a case of mitral stenosis the heart sounds are normal in the absence of murmur, though for a time the lesion may thus be quite latent.

The most common murmur present in cases of mitral stenosis is one that has no *direct* association with the lesion at all, namely, the systolic murmur of mitral incompetence, but the very fact of the presence of stenosis implies that the mitral curtains are deformed, and it is hardly to be wondered at, if the murmur significant of incompetence should be—as it unquestionably is—the most common. This murmur may replace the first sound altogether, or it may follow the first sound, modified or normal. In either case, auscultation at the back, about the region of the angle of the left scapula, may afford a certain amount of evidence in favour of the lesion, by showing that the first *sound* is distinct in this region, while the *murmur* is absent. Unfortunately, the murmur being well heard at the back, and the sound being quite inaudible there,—whether present or not at the apex,—will not enable us to exclude mitral stenosis from the diagnosis. Moreover, in cases of mitral incompetence due to simple muscle failure, the non-conduction of the murmur to the back, and the presence of the first sound at the back, are as common as in mitral stenosis.

In a statistical inquiry as to the frequency of the different murmurs in a group of sixty cases, it was found that systolic murmur was present at the apex in 75 per cent of the cases, and that this murmur was inaudible at the back in 46·66 per cent, while it was present at both apex and back in 28·33 per cent (*Medical Chronicle*, vol. iii. p. 409, 1895).

Presystolic Murmur.—In the whole wide range of cardiac auscultation, the murmur that at once has the greatest diagnostic value, and shows the greatest caprice as regards its presence or absence at different times, is the notorious auricular systolic or presystolic mitral murmur. No other murmur, besides, has such definite characters, and no other murmur depends on such an unalterable condition of lesion. The presystolic murmur has usually a definite and limited localisation—the "apex-beat"—and it is only exceptionally heard widely distributed over the cardiac area, and only very rarely is it heard at the back. The accentuated first sound, however, which may be regarded as an integral part of the murmur, is often widely conducted. As regards its special characters the presystolic murmur is absolutely different from any other murmur known in clinical medicine. It is *crescendo*, and closes abruptly at its maximum with the modified first sound referred to. The coarseness of its vibrations gives it another peculiarity, so that it is the most palpable of murmurs, while the tactile sensations it occasions are similar to the aural, in that the crescendo character and the abrupt termination, when it is at its maximum, are as pronounced in the one case as in the other. The characters referred to are so peculiar that one can hardly help associating them with the commonly accepted view of the mode of causation of the murmur by the current of blood passing through the constricted orifice by virtue of the contraction of the left auricle, which must gather force as the auricle diminishes in capacity—contracts down on its contents—and must be brought instantly to a standstill the moment the left ventricle passes into contraction.

In the statistical inquiry already referred to, the presystolic murmur occurred in 53·3 per cent of the cases; that is to say, a presystolic murmur had been heard at one time or another while the patient was under observation in this percentage of cases.

Diastolic Murmurs.—A considerably more frequent murmur in mitral stenosis is the diastolic, but its diagnostic value is much less, inasmuch as aortic murmurs of the same rhythm

are commonly well heard in the same area, and cases of simple dilatation of the heart presenting such murmur without any valve lesion are known, though very exceptionally, to occur. Even when mitral stenosis exists, and there is aortic incompetence present as well, it may be a matter of impossibility to apportion the diastolic murmur heard at the apex and over the ventricles to each of the lesions, for the diastolic murmur of mitral stenosis is much wider in its possible distribution than the presystolic, whose wide propagation is altogether exceptional.

Admitting, as we must, that aortic incompetence is frequently associated with mitral stenosis, and that its murmur is apt to be confused with the diastolic murmur of the latter, that the distribution of the two murmurs may not distinguish them, and that a diastolic murmur may be present in the mitral area when there is neither the one lesion nor the other present, our difficulties with regard to the significance of diastolic murmur are not yet at an end, although what remains of them concerns essentially the recognition or rejection of a possibly associated aortic incompetence.

The diastolic murmur that has yet to be considered has its place of distribution to the left of the sternum between the third and fourth or fifth cartilages, and is usually soft and blowing, while it immediately follows a greatly accentuated second sound. This murmur, which we have called "the murmur of high pressure in the pulmonary artery," unquestionably occurs in cases in which there has long been excessive pressure in that artery, so that its dilatation is accomplished and incompetence of its valves results, in the same way as aortic incompetence results from like changes in the aorta. In no lesion is the blood-pressure in the pulmonary artery apt to be long maintained at such a degree as in mitral stenosis, and hence the frequency of occurrence of this murmur in cases of the lesion.

At times the true diastolic murmur of mitral stenosis is practically indistinguishable from that of aortic incompetence, and is preceded by the systolic murmurs of coexisting mitral incompetence, etc., so that a double to-and-fro systolic and diastolic murmur is heard over the whole heart. In one case of the writer's, the patient came into hospital with a systolic murmur only, but later developed a loud and prolonged diastolic murmur which was widely propagated all over the cardiac surface. This murmur, along with the accompanying systolic one, closely simulated the ordinary "double aortic" murmur of aortic incompetence, except in one curious and interesting respect, that it did not seem to run off directly from the second sound, where that sound could be heard along with the murmurs towards the base of the heart. Difficult as it is to explain the intervention of an appreciable pause or interval between the second sound and the diastolic murmur of mitral stenosis, it seems impossible to understand how a pause could occur between the sound and following murmur, when the latter is due to incompetence of the arterial valves—aortic or pulmonary.

The Pulse in Mitral Stenosis.—Much discussion has taken place with regard to the state of the pulse in mitral stenosis, some observers contending that it is characterised by perfect regularity and well-maintained—even high—arterial tension, others that irregularity is its great feature. The writer has carefully studied the subject and has arrived at the following conclusions:—(1) Regular pulses of both high and low tension are commonly met with, the former

Fig. 5.—Typical pulse of mitral stenosis in first stage. Pulse regular and of good tension.

Fig. 6.—Typical pulse in mitral stenosis in second or irregular stage. The curves may be regarded as constituted of a mixture in varying proportion of those composing the pulse of the first and third stages.

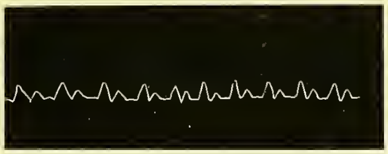


Fig. 7.—Pulse in mitral stenosis in third stage. Curves regular and of low tension.

occurring in the earlier stages of the case, the latter towards the end of the case, but low tension may be assumed by the pulse in the early stages, determined by physiological individual peculiarity or by disturbing agencies, such as the presence of pyrexia. (2) The pulse that is most characteristic of mitral stenosis is an irregular one, in which some of the beats are of fairly high arterial tension, and others abortive and of quite low arterial tension; these two types of pulse or—speaking with reference to the sphygmogram—of curve occur in all degrees of proportion, and series of beats of one or other kind are commonly interrupted by a beat or more of the other type. One may, in fact, regard this stage as made up in varying proportion of the preceding and succeeding stages already referred to. It must be admitted, however, that an indistinguishable pulse is

frequently met with in cases of simple muscle failure of the heart. (3) As the pulse was regular and of good tension in the first stage, so it may pass into a third stage in which it is again regular but of low tension.

The bigeminal pulse is not very rare in mitral stenosis, but is generally—not always—met with when the heart is under the influence of digitalis. It probably bears relation to the muscle of the heart rather than to the lesion.

There is no form of sphygmogram that, alone, enables the observer to make the diagnosis of mitral stenosis; the pulse may be regular and of high tension, it may be regular and of low tension, and it may be irregular and composed of both high and low tension beats, the latter being usually more or less abortive. Lastly, a patient quite early in the clinical evolution of the disease may have a low tension regular pulse under a disturbing influence, and as he recovers, his pulse may ultimately assume the characters of a high tension pulse, having previously passed through an irregular stage in which beats of both high and low type—the latter more or less abortive—occur side by side, though commonly the beats of one or other type appear in series, separated by the interpolation of one or more beats of the other type. Too much importance must not, therefore, be attached to the condition of pulse in mitral stenosis; it may be a "third stage" pulse when the patient is in no danger, and it may be a "first stage" regular pulse, *i.e.* of well-maintained tension, almost to the end, while the irregular or middle stage pulse is by itself of no gravity in prognosis.

Mitral Incompetence.—In no department of clinical medicine has a greater change in thought taken place during the last quarter of a century than in that concerning incompetence of the mitral valves. The importance of distinguishing *mitral disease* associated with *incompetence* and *mitral incompetence* independent of *mitral disease* at the beginning of that time was to a large extent, if not altogether, ignored. For such a state of thought the responsibility may be attributed to the ignoring of two simple, self-evident facts: (1) That the mitral curtains were never constructed to act independently of the contractile heart muscle, so that whenever the latter fails in its two obvious functions of diminishing the orifice to be closed by the valves and of supporting the curtains in action there must be a likelihood of incompetence resulting; and (2) That the great *lesion* of the mitral curtains found after death in the most common cases, the rheumatic, was not crippling of the valves, so that they could not close, but deformity of them resulting in narrowing of the orifice—in stenosis. It is not denied that lesions of the mitral curtains without stenosis are met with, amply sufficient to explain their incompetence alone, but it is affirmed that such cases are the exception and not the rule—it may be said the exception that proves the rule. Nay, in the most pronounced of them there are the best of reasons for believing that the damage of the structure of the curtains was not—even in these extreme cases—the *only* cause of the failure in the function of the curtains, but was inseparably associated in the accomplishment of such failure with weakening and debility of the heart muscle. Moreover, when stenosis of the mitral orifice was found after death in cases in which the clinical evidence for long had pointed to incompetence as the only condition, the obvious fact that whenever there is stenosis of the mitral orifice there must be deformity of the curtains, and consequently a likelihood of incompetence, was seized to do duty in explanation of the clinical experience. To put the matter shortly, the essential lesion (the stenosis) was ignored, and the mere accident of the lesion (the incompetence) was yielded a share of importance in disturbing the circulation to which it had absolutely no title. That the murmur of incompetence is by far the most common murmur in cases of mitral stenosis has been elsewhere demonstrated, but it is idle to say that the minute regurgitant current it probably denotes is the cause of the profound disturbance of the general circulation in mitral stenosis.

The cases associated with the most serious damage to the valve curtains, apart from stenosis, are those of septic endocarditis, in which the disturbance of the general circulation that must inevitably result is but a minor matter in the evolution of the case, seeing that hitherto the disease has proved inevitably fatal in an acute or at least subacute illness, which compels the patient to remain in bed (*vide* "Septic Endocarditis," p. 113).

Skoda, it is believed, first called attention to the frequency of a systolic murmur in the "pulmonary area" in association with mitral incompetence and the consequent dilatation of the right side of the heart, and many years later the view (which we cannot regard other than as a great heresy) was advocated that this systolic murmur in the "pulmonary area" was nothing else than the murmur of mitral incompetence conducted to the surface at this spot by the left auricular appendix presumed to be dilated. In support of this theory a disposition was manifested to change the position of this long-familiar murmur, and move it outwards "half an inch or more to the left of the sternum" in the second intercostal space, to the spot corresponding to the tip of the left auricle as it lies to the left of the pulmonary artery. It was alleged that in chronic cardiac cases associated with habitual engorgement of the right side of the heart, the appendix of the left auricle becomes dilated, and to such an extent, indeed, as at times to occasion visible pulsation

in the second interspace. Moreover, it was held that the murmur of mitral incompetence is frequently heard over such dilated appendix, conducted as if by predilection to this spot even when absent at the cardiac apex! The pulsation in the second left intercostal space referred to, used to be regarded as auricular systolic to fit in with this explanation of its being due to the auricular appendix, but as a matter of fact this rhythm of impulse could not be demonstrated — the impulse was obviously systolic. Worsted in their argument, the advocates of the auricle being the seat of the pulsation in the second left space shifted their ground, and contended that the pulsation and the murmur were alike due to a regurgitant current through the mitral orifice from ventricle to auricular appendix!—the deviousness of its course being completely ignored.

As a matter of fact, in all cases of cardiac disease in which the obstruction tells heavily on the pulmonary circulation, and in which the right chambers of the heart become enlarged, the right ventricle rather than the appendix of the left auricle it is that bears the brunt of the struggle and is the first to become enlarged. The advocates of the view we are considering would have had a less unfortunate case had they limited their theory to cases of mitral stenosis, in which the auricle is apt to be dilated, though its appendix may take no part in the change and be actually blocked with clot, but the great field of anæmic and other cardiac debilities was too tempting for them. Henceforth was not the notoriously common murmur of anæmia explained to their satisfaction? They had evidence of general cardiac disability in plenty, but why should such special disability fall thus to the share of the left auricle so as to account for its disproportionate dilatation in the absence of mitral stenosis? As we have affirmed, as a matter of fact post-mortem examination demonstrates, that when there is obstruction in the pulmonary circuit it is the right chambers of the heart that dilate, and that when there is obstruction at the mitral orifice the same chambers suffer the like change, though in common with the weak-walled left auricle, whose appendix suffers least, or even not at all. When, however, the right ventricle becomes dilated its infundibular portion is especially prone to give way, and as the conus dilates it tends to thrust aside the appendix of the auricle, while the position of this chamber proper is essentially a posterior one—very far back indeed. The pulsation attributed to the left auricular appendix is obviously the pulsation of the infundibulum, as was long ago shown by Dr. Mahomed. Then, again, whenever there is obstruction in the pulmonary circuit of such intensity and duration as to occasion pulsation in the second left interspace, close to the sternum, it is usually easy to feel with the hand the shock due to the too forcible closure of the semilunar valves, because they are here placed so superficially.

It will be asked why in the most common cases—the anæmic—the right chambers of the heart should specially suffer? The cause is probably that the morbid blood has difficulty in passing through the lungs; whether we cut short the supply of oxygen from the respiratory passages, or limit the capacity of the field for oxygenation, must not the result be similar? and if so, must not embarrassment of the right chambers be the first result—that of the left being often considerably postponed? In severe fevers in which the tendency of the blood to stagnate in the lungs is notorious, Stokes long ago called attention to the engorgement of the right ventricle, whose impulse grows apparently stronger as that of the left ventricle fails.

But though so well developed in cases of anæmia this systolic murmur in the pulmonary area is a not rare one in all kinds of cardiac disability, and in them too it may stand alone—the only murmur present in the whole cardiac area. The murmur, again, is not very rarely met with in the examination of candidates for assurance and for the public services, who seem to be in perfect health—possibly some anatomical abnormality of contour or course may be the explanation. We have already called attention to the dilatation of the pulmonary artery itself, that is met with in cases of long-standing mitral stenosis and other conditions resulting in congestion in the pulmonary circulation, and referred to the dilatation and actual atheromatous change that may ensue in a vessel so exposed to excessive pressure. There seems to be no reason why a systolic murmur should not be produced in the pulmonary artery under these circumstances, in the same way as under like circumstances it is produced in the aorta. Most of those who have been engaged in teaching clinical medicine have probably at one time or another met with this experience in the presence of an inquiring student:—A patient with a first attack of acute rheumatism develops a systolic murmur at the apex, but in a day or two there may be a similar murmur in the other areas, including the tricuspid and pulmonary; "Why," asks the inquiring student, "am I told that the same murmur which indicates disease of the valve in the mitral area has no such significance in the tricuspid and pulmonary areas?" The teacher will do well to base his reply on our accumulated clinical and pathological experience of facts, and not attempt a reply based on any theory. That such a murmur in the pulmonary area is common under the circumstance is unquestionable.

It must be admitted that as yet we have little knowledge of the mode of production of a systolic murmur in the pulmonary area, either standing alone or associated with murmurs else-

where: it is probable, too, that the mode of production is not always one and the same. In a case of evident muscle failure of the heart—say due to alcoholism—this murmur may be the only murmur present, and it is difficult to deny its definite association with the muscle failure and consequent cardiac disability. No one will deny the element of muscle failure in most cases of anæmia. The murmur as an entirely isolated phenomenon, as met with, for instance, in apparently healthy candidates for the public services, is more difficult of interpretation. The practical rule, however, must be that a systolic murmur in the pulmonary area is the murmur of lowest value among all the murmurs with which clinical practice has made us familiar. Occurring in a case of evident muscle failure, it adds little information to that already acquired in other directions, but on the other hand one must not neglect the murmur altogether, seeing that it is not rarely associated with conditions of cardiac disability.

Mitral Incompetence as a Result of Muscle Failure.—One of the most common results of muscle failure of the heart—and this quite independently of its pathological cause—is mitral incompetence that is in no way associated with any lesion of the valves. Mitral incompetence of this mechanism is met with under many morbid conditions, of which we will take only two as examples: the heart failure of anæmia and the heart failure of Bright's disease—the one curable, the other sooner or later lethal from concomitant circumstances, though capable of great and prolonged improvement from time to time during its course. The murmur of mitral incompetence present in the former cases was for long misinterpreted, while that present in the latter cases was for as long practically ignored, though the obvious signs of a failing heart were only too striking in the forms of dyspnœa, dropsy, and engorged liver, not to speak of the distension and pulsation of the veins of the neck and a host of minor indications.

Physiologists a long time ago recognised what they termed "the safety-valve" action of the tricuspid apparatus, by which term they denoted the fact that under excess of work to be done by the contraction of the right ventricle, the healthy tricuspid valves may permit regurgitation taking place, and so relieve for the moment embarrassment of the ventricle. It is known now that the same occurrence is common, and of much greater importance on the left side of the heart. When the arterial pressure is high, and the left ventricle labouring to maintain the circulation, it is now a familiar fact that the mitral valves often become incompetent, and that, too, in all probability for the immediate if not for the permanent benefit of the individual.

Muscle Failure of the Heart.—The consideration of mitral incompetence necessarily leads us on to the consideration of muscle failure of the heart in general, of which it is so often a secondary consequence. Probably in no case of mitral incompetence can it be said, however grave the structural damage to the curtains, that either the incompetence or the disturbance of the general circulation is solely the result of such structural damage, for invariably associated with this latter is an element of muscle failure that we cannot possibly afford to ignore. But there are cases of muscle failure that run their course and display all the ordinary indications of a failing circulation without ever developing, as far as we know, the condition of mitral incompetence. Of a silent mitral incompetence we know nothing, and considering the great frequency of the condition as denoted by its murmur, it seems likely that a silent regurgitation through the mitral curtains is a pure assumption having no basis in fact.

How then is to be explained the unquestionable occurrence of venous stasis and its usual effects as the result of muscle failure of the heart without any evidence of mitral incompetence from first to last of a long course? Evidently the explanation of the fact is amply provided for by the clearly established fact that an overburdened ventricle—be it right or left—has another mode of expressing its embarrassment than that obtained by the permission of incompetence of its auriculo-ventricular valves. The occurrence was long ago recognised (only an unfortunate name—asystole—naturally associated with most lethal significance, was given it, and the frequency of its establishment ignored), and consists of *the non-completion of systole.* In health the supra-papillary space of the ventricle, *i.e.* immediately below the valves, always contains blood at the end of systole, but with this exception the chamber is normally emptied. When there is muscle failure, however, residual blood may remain at the end of systole elsewhere than in the supra-papillary space. If any one has doubt as to the truth of this statement, let him look at the heart post-mortem in a case of muscle failure with huge rounded dilatation of the left ventricle, and ask himself how long he can suppose it is since such a ventricle completed its systole in the physiological way. To denote this condition of incomplete systole, on the advice of a scholarly friend, the writer has used the term *systole catalectic*, implying that the systole stops short of its completion.

One would naturally suppose that the same muscle failure that occasions systole catalectic would also occasion mitral incompetence, but as a matter of clinical experience this does not seem to be necessarily the case, though the two conditions are no doubt commonly coincident.

There are two auscultatory signs of very different nature, that are so commonly associated with muscle failure of the heart as evidently to bear definite relationship to it: one of these is so frequently met with under other circumstances

—mitral valve disease and mitral stenosis—as to be little characteristic, the other we may consider as the *special sign of muscle failure* whether the latter be transient or permanent. The former is the murmur of regurgitation through the mitral and tricuspid orifices, owing to incompetence of the sound mitral and tricuspid valves, but this has been already referred to in sufficient detail.

Till lately the possibility of a diastolic murmur being produced in a simply dilated heart, the result of muscle failure, was usually emphatically denied, but of recent years evidence has been accumulating to show that a diastolic murmur of very considerable loudness (and actually accompanied by a thrill) may be produced in a simply dilated heart without any valve lesion whatsoever. In the face of this recent experience it is curious to find recorded in Dr. Stokes' classical work on Heart Disease, published in 1854, a case of the kind. Two cases occurred in the Manchester Royal Infirmary a few years ago, and were recorded in the *Practitioner* for 1894. In these cases not only was there a diastolic murmur (accompanied by thrill), but there was also a reduplicate diastolic sound at the apex. During life, both cases had been regarded as cases of mitral stenosis, but neither this lesion nor aortic incompetence could be found post-mortem. It is sufficient here to point out the existence of such cases, however rare they may be. So seldom are they met with, that for practical purposes they scarcely impugn the usually accepted rule that a diastolic murmur means either arterial incompetence or auriculo-ventricular stenosis. They are of immense importance, however, in demonstrating to us how profoundly ignorant we are as to the mode of production of some of the signs with which we are well acquainted. The diastolic murmur referred to is of extreme rarity, but the *bruit de galop* (to be immediately described) is one of the most common of auscultatory signs, yet as to the mode of production of both these signs no satisfactory explanation has ever been given.

The auscultatory sign we are fain to regard as the great sign of muscle failure is a peculiar triple rhythm of (apparently) the cardiac *sounds*. The sign attracted the attention of French physicians long before it received from English authors the consideration it unquestionably merits, and so the French name has almost passed into our own language, and we call the sign the *bruit de galop*. Evidently one of the sounds is systolic and two are diastolic. Indeed, as already noted, there may be felt at the cardiac apex two distinct shocks or thuds that correspond with the diastolic sounds. We have, perhaps not without some clumsy writing, avoided the use of the term "reduplicated second sound," which might seem the natural one to employ, because of our belief that the diastolic sounds do not represent the closure of the semilunar valves. We might go a step farther, and say that in our belief neither represents the physiological second sound, and thus express our scepticism of theories that assume an interpolated sound to be the cause of the triple rhythm. The true *bruit de galop* is a sign always best heard over the ventricles, *i.e.* over the chief part of the muscle of the heart, and though occasionally it may be heard widely distributed and even audible at the base, in the latter situation it is less loud than over the body of the heart. The *bruit de galop* may be heard in all forms of muscle failure of the heart—even in that due to anæmia—but the most perfect examples are supplied by cases of chronic Bright's disease, when the vigour of the hypertrophied heart muscle is on the wane, and so onwards to the end. Often it is associated at one time or another or permanently with the murmur of mitral and tricuspid incompetence, but its most perfect development occurs in the absence of any murmur, and its importance as an auscultatory sign perhaps surpasses—or at least is equal to—that of any murmur, its therapeutic bearings are so significant. It *means a failing heart muscle.*

The Pulse in Muscle Failure of the Heart

The condition of the pulse in cardiac muscle failure is one of very great importance, inasmuch as it forms the guide to the method of our therapeutic endeavour. *All degrees of arterial tension may be associated with muscle failure from the highest to the lowest*, though the former cannot be expected to be long maintained in the presence of weakening of the muscle of the heart. Moreover, intermittence, inequality of beats, and all forms of irregularity up to the fancifully named "delirium cordis" pulse, are quite commonly met with. Tachycardia, too, may occur, and cannot be regarded without apprehension, seeing how it must wear out a heart when long continued. The opposite condition, bradycardia, may likewise be encountered.

It cannot be too strongly insisted on that failing heart and high arterial tension are often associated together; nay, the latter is a powerful agent in the determination of the former. The combination is often witnessed in the course of chronic Bright's disease, and during the association it is that the most distressing dyspnœa is witnessed, a struggle, as it were, being maintained between heart and vessel.

As the heart muscle fails, however, the tension tends to fall, as it might naturally be expected to do under the circumstances, and it may ultimately reach a very low grade, the tidal wave of the tracing disappearing altogether, and dicrotism becoming pronounced. The presence or absence of mitral incompetence seems, however, to be in no way indicated in the sphygmogram. Irregularity is common, especi-

ally in the form of sphygmogram already described as that of the second stage pulse of mitral stenosis. But often an earlier change is occasional simple intermission. Almost as frequently there is an abortive beat represented in the sphygmogram during the intermission of the pulse that is felt at the wrist. This is the so-called "missed beat." Lately the writer met with a case which, though the intermission as felt at the wrist was only very occasional, invariably *two* in place of *one* beat proved abortive—in fact there was a rare *trigeminal* beat. A continuous trigeminal pulse is rare for any length of time, but not so a bigeminal one, and though often associated with the effects of digitalis, it is by no means always so. Whenever an apparently slow pulse is felt, the possibility of there being an intermediate beat imperceptible through the finger—in other words, of our having to do with a bigeminal instead of a bradycardiac pulse—must be entertained. Examination of the heart with the stethoscope will at once indicate whether this is the case or not. The heart sounds, however, are most difficult to analyse in the presence of this peculiar action of the heart, and the writer questions the utility of entering upon a discussion of the subject. Of course murmurs are often present and add to the complexity of the combination, which may be met with in any kind of heart disease, and would seem, therefore, to be directly associated with the state of the muscle of the heart and of its nervous mechanism.

True bradycardia or slow pulse is a rather rare condition, and may be, as far as we know, congenital. It is remarkable, as we shall find, inasmuch as its effects are observed specially on the arterial side of the circulation, syncopal or epileptiform attacks being liable to result from cerebral anæmia (*vide* p. 154).

It is evident from the foregoing remarks that several quite distinct types of pulse may be met with in cases of simple muscle failure of the heart, and the variations they present may be extreme; thus there may be very high tension in the struggle against which the heart muscle is beginning to fail, and there may be extremely low tension when the heart has utterly failed or there is general vaso-motor paralysis. There may be, again, bradycardia or tachycardia and all forms of irregularity, varying from a rare intermission to a state of utter disorder in which it is impossible to recognise any prevailing type or types of curve. It follows from these remarks that from a diagnostic point of view little is to be expected from observation of the pulse in muscle failure, but it is far otherwise when therapeutic indications are sought in the same direction.

Treatment

The treatment of heart disease includes much more than the mere administration of drugs, powerful for good or ill though these may be. Nothing seems more rational than the enforcement of *rest* upon the cardiac sufferer. It is impossible to give his heart rest; all we can aim at is diminishing its work as far as in us lies. Of late years much has been heard of a treatment in which graduated exercise plays an essential part. This method of treatment is fully described elsewhere in this work (*see* Therapeutics, Physical), and it is not for the writer to express his opinion upon it, but he may be permitted to quote from Dr. Stokes' work on Heart Disease, published in 1854, the following passage to show that, as far as the exercise is concerned, the treatment is not of such recent origin as is often supposed. The patient is enjoined by Dr. Stokes "to pursue a system of graduated muscular exercise; and it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise with pleasure and advantage which at first was totally impossible owing to the difficulty of breathing which followed exertion. . . . The symptoms of debility of the heart are often removable by a regulated course of gymnastics or by pedestrian exercise, even in mountainous countries such as Switzerland or the Highlands of Scotland or Ireland." The term "debility of the heart," used by Dr. Stokes, is a singularly appropriate one, and the condition for which the treatment can be alone regarded by the writer as appropriate must be something resembling that present in the sedentary man, who,

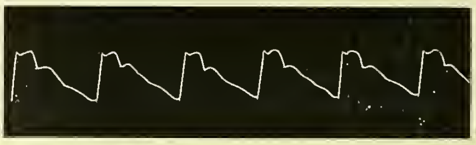


Fig. 8.—High tension pulse in case of Bright's disease associated with mitral incompetence resulting from a degree of muscle failure of the heart. Man æt. 38.

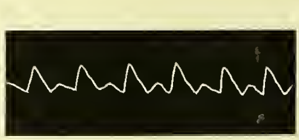


Fig. 9.—Low tension pulse. Mitral incompetence associated with endocarditis and adherent pericardium. Man æt. 25, who had rheumatic fever a year before tracing was taken.

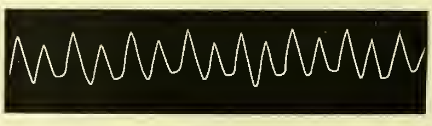


Fig. 10.—Extremely low tension pulse in septic endocarditis. Mitral incompetence associated with much damage to valves. The great size of the dicrotic wave is noteworthy, and the fact possibly bears relation to the pyrexia present.

besides being often an injudicious feeder, allows his whole muscular system, including his heart, to become "flabby." This is all the reference

FIG. 11.—Muscle failure in chronic Bright's disease (granular kidney, p.m.). Man æt. 47, who had at the time no incompetence of his mitral valves, and who recovered from the symptoms of cardiac failure, and lived for nearly ten years.

FIG. 12.—Tracing from same patient towards the end of convalescence during first stay in hospital; pulse of good almost high tension.

FIG. 13.—Temporary great irregularity of the pulse. "Delirium cordis." From a case of muscle failure of heart in a case of Bright's disease (gran. kidney).

FIG. 14.—Usual pulse of same patient.

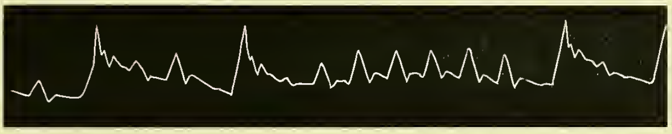


FIG. 15.—Irregular pulse in the cardiac muscle failure of Bright's disease (gran. kidney). Patient from whom Figs. 11 and 12 were taken, but towards the end of the case. Mitral incompetence.

FIG. 16.—Intermission of the pulse. The long upstroke of the curve that follows the intermission is to be noted.

FIG. 17.—Bigeminal pulse in fatal case of alcoholic muscle failure of the heart.

FIG. 18.—Alternating pulse in muscle failure of the heart.

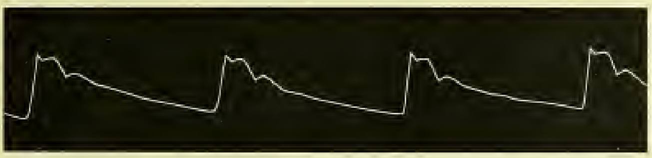


FIG. 19.—Pulse in case of typical bradycardia associated with syncopal seizures.

FIG. 20.—Tracing of pulse taken during an attack of tachycardia. Diabetic patient without obvious sign of cardiac disease.

that the writer will make to the subject, but he takes the opportunity of reminding the reader of the immense influence exerted by exercise and careful feeding in the process of athletic training, in which process no one can doubt that the heart is largely influenced—without exaggeration one may say *chiefly* influenced, seeing that it is "wind," so closely related to the heart's health, that dominates the successful result of the process. Without good "wind" dependent on a vigorous heart muscle the most perfect development of all the other muscles of the body would be rendered useless. But the fact is too obvious to require statement.

It will be generally conceded that if a man has an aneurysm of his left ventricle, the result of an obliterated coronary branch, or if one or both of the main coronary vessels are narrowed at their orifices by chronic aortitis, nothing but disaster could follow his submission to treatment by exercise. Valve defect, however, is an intensely mechanical result of disease in the first instance, and the muscle of the heart may remain for long perfectly sound in its presence, and therefore, we may say, capable of improvement in strength by the "gymnastic" process, but even in this case we must bear in mind that the heart muscle by virtue of such valve defect is always being, as it were, *exercised to the full*, and that it will not thus have the usual margin for improvement in strength. There is no valve lesion that does not throw strain upon some part of the heart muscle, and the "solidarity" of the organ must be remembered. When a patient comes seeking treatment for heart disease, it may be granted that he has, at least, the first of the cardinal symptoms—dyspnœa on exertion. Already, then, the circulation is disturbed, and the heart has begun to be overborne in the struggle. *Rest*, with the object of relieving the heart of its work, as far as that is possible, is now imperative, and *rest alone* will often suffice to turn the balance in favour of the heart, so that the circulation is restored and the disorder of functions, resulting from its disturbance, disappears. No doubt a large number of patients, who have all the three cardinal symptoms of heart disease and many subordinate ones, would recover with a profuse diuresis, unaided by any treatment by drugs, if it were the custom (and we are glad it is not) to withhold medicinal treatment for several days. The fact of some patients doing so, however, teaches an emphatic lesson as to the importance of rest, which should be absolute in order to yield its greatest benefits. Patients in advanced stages of heart disease will often say that rest in bed is impossible for them; this is true in a few cases, but only in a few. Patients, however, are often unable to lie down in bed (orthopnœa), but this is a much less important matter. When a patient will only sit in a chair, often leaning on the back of another chair or other piece of furniture, his dropsical legs are not likely to be relieved unless it be by acupuncture: on the contrary, their condition is apt to be aggravated, and the tense skin may ultimately crack and exude serum, while an erythematous condition is apt to supervene, which rarely eventuates in sloughing. It is very desirable, then, that the sufferer from heart disease should remain in bed, though it is astonishing how frequently bed is energetically resisted by the patients who require it most. In hospital practice we find, however, that the number of patients who really cannot stay in bed owing to their dyspnœa is very small indeed, apparently not so much by persuasion being more successful as by force of example. The writer has reason to believe that the imperative necessity of *rest in bed* is often not sufficiently insisted on by the private practitioner; that is to say, the patient's consent is not obtained when it easily might be. One of the dangers of heavily narcotising a patient with severe heart disease is his being suffocated by mechanical interference with the upper air-passages, by bending his neck too acutely, or letting his head press upon folded arms and so forth, and such an accident is more likely to occur when the patient sits up in a chair. In the few cases referred to in which the patient *must* sit up, this necessity will often disappear in the course of a few days, during which medicinal treatment effects amelioration. The form of dyspnœa called "Cheyne-Stokes respiration" is often associated with distressing restlessness during the dyspnœal paroxysm, the patient often actually springing out of bed during the height of the seizure, a condition that seriously militates against the chances of recovery, otherwise small enough. Active delirium, again, is a serious symptom on its own account for the same reason; but both Cheyne-Stokes respiration and delirium are symptoms for the most part met with only in the advanced stages of heart disease, and in the most unmanageable forms of the disease, as that sequential to chronic Bright's disease.

In conclusion of the subject of rest in the treatment of heart disease, the writer would emphasise the importance of a careful general survey of the whole case in determining the amount of rest to be enjoined, and especially, when that amount has been obtained, the importance of a *gradual* resumption of activity. All the benefit derivable from the rest may be flung away by injudicious management in this respect at the end. Nay, fatal syncope may be induced by the patient being *suddenly* allowed to assume his wonted activity. But short of such an accident the faith of the patient in the efficacy of his enforced rest may be so shaken that he will never again submit to rest while he has the power of resisting it.

The Weir-Mitchell treatment has always seemed to the writer a method of accomplishing

rest with the great advantage of abundant nourishment at the same time being rendered innocuous in spite of the denial of voluntary activity to the muscles. The muscles are "exercised," as it were, by the masseur—that is to say, are helped to the disposal and removal of their often toxic products of metabolism—without any strain on the heart. Rather, we might fancy, does the process actually relieve the heart by rendering the peripheral circulation more easy. Of course, in the presence of much general dropsy, the kneading of the muscles is rendered impossible; but it is not under these circumstances that the question of this treatment is likely to arise.

Diet

We have next to consider that most important subject in the treatment of heart disease—diet. That this subject is too often ignored by the medical practitioner is abundantly evident by the frequent bitter complaint by the patient of his gaseously distended abdomen, which, pressing up on his diaphragm and impeding his already embarrassed respiration, occasions him infinite discomfort. If there is some albumin in his urine, as is the rule in most severe cases, so much the more is the patient likely to be injuriously fed with excess of carbohydrates, rice, tapioca, and the like pernicious puddings, while his nitrogenous food is cut down to a minimum. If we reflect that the heart is a muscle, and the most important muscle—not to say the most important organ—in the body, surely we ought to give the food that long experience has taught us is best for muscle tissue—nitrogenous, and not carbohydrate articles. The modern system of athletic training is, the writer understands, less strict in this respect than that of former days, but he doubts if the "staying powers" of its subjects are any better in consequence—if as good. However, past records are good enough for him. The patient with severe heart disease cannot take exercise, and we must recognise the fact. His muscles must lie fallow, except in the very few cases in which general massage is adopted. But is the case for excess of carbohydrate food any better on that account than that for a preponderance of nitrogenous food? If the physician fears an excessively nitrogenous diet, let him reflect that the toxic nitrogenous bodies in the blood he fears are promoted, not diminished, by a largely carbohydrate diet, inasmuch as the articles of the latter will use up the oxygen necessary for the metabolism of the tissues and for dealing with its nitrogenous products. Another consideration is the state of the liver, in all cases with venous stasis: the nitrogenous elements of food and the nitrogenous products of metabolism no doubt undergo change in the organ; but so also—and probably in greater amount—do the carbohydrates, so that the abnormal state of the gland that must result from its congestion with venous blood is at least as important in the one case as in the other. We should say that excess of carbohydrates in the diet was infinitely worse than an excess of nitrogenous elements, provided that carbohydrates are at the same time cut down to a minimum. So much for the question of metabolism. But for the primary disposal of food in the stomach what does reflection teach? A rice pudding alone is probably rapidly passed through the stomach, and does no mischief there; but far otherwise is the case if nitrogenous food, as beef or mutton, is given at the same time. Evidently the rice pudding will simply clog the process of gastric digestion by its bulk, and if digestion is weak it is only too apt to be arrested altogether, a decomposing mass of incongruous stuff being ultimately passed into the intestine to create discomfort lower down. We may start with the assumption, well borne out by the scientific labours of the late Sir William Roberts, that the healthy human being has great excess of digestive power, and can afford to conform to the customs of society in the mixing of his foods. Most healthy people can do this up to a good old age; nevertheless, in many difficulty of gastric digestion is experienced early in middle life, in which case relief can often be obtained without medicinal agency by the mere simplification of the dietary; that is to say, the patient lets his stomach's full energy be exerted on his nitrogenous food, while he is equally careful to take his carbohydrate food at a different time from that at which he takes his nitrogenous food, thereby, no doubt, promoting its speedy passage through the stomach to the parts beyond, which are capable of dealing with it, all the more that the saliva, with which it ought to be saturated, has probably to a large extent escaped being interfered with by the acid of the juice in its rapid transit through the stomach. We plead, then, in the case of cardiac sufferers, for *simplification of their food*, in the first instance, and in the second for the *reduction to a minimum of their carbohydrate food;* the object of the first principle concerning the primary digestion in the stomach, that of the second principle concerning the metabolism of the tissues. We have spoken only of the stomach, but the digestion of food and the impeded absorption of food and fluid throughout the whole tract of the alimentary canal beyond must not be forgotten in a heart case. We have dwelt in symptomatology on the distressing tympanites of heart sufferers, and in its production the important part played by food is only too patent. Far better than any drug treatment, by intestinal antiseptics, is the simplification of the alimentation of the sufferer, bearing in mind his peculiar disabilities and how all his digestive powers are handicapped by the venous stasis of his digestive

organs, which, judging from the condition of the spleen, would be still worse were it not for the ready distensibility of the liver.

Let us next inquire how these principles may be carried into effect in actual practice. The sufferer from heart disease is usually free from fever, and there is no necessity, unless he has temporary gastric catarrh, to feed him on "slops"; there is no reason, usually, why he should not retain the ordinary habits of health and have three meals a day. These should be small in bulk, easy of digestion, and arranged at sufficient intervals to permit of complete removal of one meal from the stomach before the entrance of the next into it, and of course a period of inactivity should be allowed to the organ. Long fasting, as from breakfast to late dinner, is to be deprecated. This habit is not infrequent in the earlier stages of heart disease, while the patient is still able to attend to his business. The meals should, further, be as *dry* as possible, with the exception of breakfast, and on this account only a small quantity of soup (and that clear) is permissible—but soups are not to be recommended. Half a glass of whisky or brandy in half a small tumbler of water may be allowed with the mid-day and evening meals. Breakfast should form the carbohydrate meal of the day, and may consist of thin crisp toast "done through," and not having a spongy centre, which is to be buttered cold, care being taken to have the butter of the best quality, and of a soft-boiled egg, or even two. A large cup of boiled milk, flavoured with coffee or Chinese tea, and sweetened with saxin, should constitute the fluid of the meal. If the patient craves for it, and it is found to agree, the *fat* of bacon may be allowed. The egg may be poached if preferred, or even fried. If the patient cares for it, fruit may be allowed. Eight or nine o'clock will usually be the time chosen for breakfast, and the mid-day meal will then be taken at 1 or 2 P.M. Cups of bovril and the like are to be deprecated in the forenoon. The mid-day meal should consist of a chop, fish, or fowl, with some well-cooked *green* vegetables—cabbage, spinach, and the like. Boiled cabbage may be squeezed through a sieve so as to break up its fibres, and should be cooked with plenty of butter, if this does not disagree. Reference to butter reminds the writer to allude to the not uncommon experience that certain patients are intolerant of fats, which it is difficult to get them to take, while others, who often experience a difficulty in the digestion of starchy foods, have a great capacity for consuming fats. These and other idiosyncrasies, such as inability to eat the smallest particle of an egg without suffering, must be recognised and respected when they are ascertained to be well founded. With the mid-day meal fruit in season, either cooked (saxin being added if sweetening is necessary) or in the natural state, may be allowed. Cheese is apt to prove indigestible; but some patients have a surprising facility in digesting it, and to them a small amount may be allowed along with butter, but on no account is bread or biscuit to be taken along with it. At five o'clock a cup of pure Chinese tea, sweetened with saxin, may be taken, *but no bread or biscuit on any account.* At 7 or 7.30 P.M. the evening meal is taken, and should be, in principle, practically a repetition of the mid-day meal; only, if chop has formed the latter, fish, or fowl, or a joint may constitute the former, and so on. Green vegetables are to be taken again. Patients often demur to taking green vegetables, under the impression that these increase tympanites. This is not the writer's experience when carbohydrates are at the same time withheld. There is no great objection to a double course—say, fish and a joint,—or occasionally a *small* quantity of clear soup may precede the joint, fish, or fowl, as it happens to be. Fruit may follow; cheese is perhaps better avoided. These three small meals will usually be abundance for the cardiac sufferer; more he probably could not utilise, even though he were capable of properly digesting it. The writer used to feed his patients more largely than experience has since taught him to be desirable.

A glass of whisky or brandy in half a tumbler of hot water about eleven or twelve o'clock will often help the patient to a good night, but *on no account must any biscuit or bread be taken along with it.* In the case of the so-called working classes the writer is continually contending against the terrible "tea-meal" that patients of this class are so reluctant to give up. In a class somewhat higher, this same meal is made still more noxious by some nitrogenous elements of food being thrown in—fish, chops, and the like—to form in the stomach, amid a large amount of bread and butter, and tea and jam, a mass indeed well calculated to frustrate the efforts of the most potent juice to act upon it. The writer has long abandoned the names "dinner" and "supper" for the mid-day and evening meals, which are simply so termed; he is convinced that our working classes would live healthier lives if they deferred the principal meal of the day till evening—say, till an hour after their return from work, which hour is well spent in repose. He has been told that the principal meal is requisite in the middle of the day in order to supply strength and energy for the labour of the day. The taking of a *small nutritious* meal in the middle of the day, and the reservation of the principal meal till the day's work is over, would in his opinion be a much better system, a substantial breakfast being of course requisite.

As already stated, it is astonishing how well, in many heart cases, the digestive powers are retained even in the presence of considerable

portal congestion, nay almost to the end of the case. But portal congestion unquestionably implies a proneness to catarrhal inflammation of both stomach and bowels. It is in the case of the former organ that this process is most evident—the patient losing all appetite and then vomiting his food. (The possibility of digitalis being a factor in the sickness must always be borne in mind.) For this condition medicinal treatment alone avails little. Large doses of subnitrate of bismuth constitute probably the best form of it, but in a severe attack deprivation of all food by the mouth is necessary, nutritive suppositories or enemata being given per rectum. In any case the food taken by the mouth should be limited to milk, and this is best peptonised, as there can be very little digestive power in the stomach during catarrh. Fermented milk or "koumiss" (originally mare's milk) will often be retained when ordinary milk is rejected. But it fails in a certain proportion of cases, and then there is no alternative but to let the stomach have absolute rest for a time; usually twenty-four or forty-eight hours suffice for recovery. Care, of course, must be exercised in the resumption of nourishment by the stomach—only bland fluids in small quantities at a time are at first permissible.

Mention must be made of an absolute milk diet in the treatment of heart cases, especially those associated with high arterial tension and kidney disease. This treatment is only applicable to the patient confined to bed. It would seem that the products of metabolism arising during milk diet and rest are much less toxic than those produced while ordinary diet and exercise are being taken; and as a consequence the vascular tension declines, an important matter, seeing that it directly concerns the heart. No doubt another element in the good effects of the treatment is the speedy washing away of any toxic products of metabolism there may be. A tendency to constipation can easily be obviated by laxative drugs. But few patients will or can continue the treatment long.

It is such a common practice to give frequent purgatives in heart disease, that it is usually hard to disassociate catarrh of the bowels from their action. Moreover, the mucous membrane of the intestines in a severe heart case is sure to be exposed to the venous stasis of the portal system. Notwithstanding, it is not often that diarrhœa calls for treatment, a moderate looseness being usually considered desirable. It must be remembered, however, that any approach to acuteness of the catarrhal process is apt to exert a depressing influence upon the heart; while frequent actions of the bowels imply exertion on the part of patients who do not use a bed-pan. These considerations lend additional importance to the dietetics of heart disease, and the writer believes that tympanites, so distressing to many sufferers from the disease, is largely promoted by excess of carbohydrates — the usually innocently regarded rice pudding and its kindred—while well-boiled green vegetables and even fruit may be taken with impunity provided they are taken with nitrogenous food alone. It is a remarkable fact that cases of obstinate chronic tropical dysentery after resisting all medicinal treatment are sometimes cured expeditiously and permanently by the consumption of large quantities of fruit, apples, etc. The permission to take fruit may seem to be contrary to the principles of diet already laid down, but a patient, whose starchy food and cane-sugar is as much reduced as has been advised in heart cases, can consume the glucose of fruit with as much impunity as the patient on milk diet consumes his lactose. We are not dealing with diabetic patients, though an apparent sugar reaction is sometimes obtained in the urine of cardiac patients; generally it is due to excess of uric acid, or the presence of some abnormal body, as glycuronic acid; sometimes really to glucose. When we consider the disorder of the liver that must result from heart disease, it is surprising rather that glycosuria should be so rare than that it should occur occasionally in the urine.

Medicinal Treatment.—We have next to consider in detail the treatment of heart disease by drugs. The powerful and toxic remedies that act directly on the heart muscle itself must of necessity have the first place in the consideration of these remedies. One may be said to be *facile princeps*—a giant among the rest—namely, digitalis, which, take it all and all, has no rival. A knowledge of the peculiarities of action of this remedy, which include the slowness of the establishment of its diuretic effects, its cumulative properties, by virtue of which its action is maintained for a considerable time after its administration has ceased, and its double, in a sense antagonistic action, at the same time on both the heart muscle and the peripheral arterioles, is of immense importance in the employment of digitalis in practice. The effect desired is a tonic one on the heart muscle, the best indication of the achievement of which is an increase in the flow of urine steadily maintained for several days. And once for all, let it be clearly understood that it is the *heart muscle*, and not any *lesion* there may be, that is influenced by the drug. The writer has absolutely no faith in the suitableness of the remedy in cases of one lesion, and in its unsuitableness in cases of another lesion. Such views, he thinks, have sadly often led to the withholding of digitalis when it constituted the only hope of the redemption of the case. Aortic incompetence and mitral stenosis are the two lesions in regard to which purely theoretical considerations have been allowed to exert such a grievous influence on practice.

With regard to the former, the writer can only endorse the view of his old master, Dr. G. W. Balfour, and maintain that aortic incompetence, far from affording cases that are unsuitable for the administration of digitalis, supplies the most brilliant examples of its curative effects. He would go further, and add that on calm reflection theory itself bears out the actual clinical experience of the treatment of cases of the kind by digitalis. Aortic incompetence is the lesion of all others whose effects fall directly upon the left ventricle, the most powerful part of the heart muscle, and when we give digitalis under the circumstances we get its effects exerted in chief force just where it is most wanted. In mitral stenosis, a better case for the view he has condemned the writer admits can be made out; for in its case we have no powerful muscle wall in the chamber first concerned to influence and stimulate. Before we can bring increased muscle vigour to bear on the obstruction the pulmonary circulation must be congested, and after all, the right ventricle is at best wretchedly weak in comparison with the left ventricle. Moreover, in the earlier stages of the lesion the pulse, as we have found, is one of considerable tension, the sphygmogram displaying a well-developed tidal wave. But practical experience overrides these considerations. The study of cases of mitral stenosis to their end brings fresh strength to the belief in the practical usefulness of digitalis, for in the vast majority of cases the *left ventricle is enlarged.* When this unquestionable fact is explained in as unquestionable a manner, the writer is prepared to reconsider his position in regard to the efficacy or harmfulness (for if it does not do good, so powerful a remedy cannot be simply inoperative) of digitalis in mitral stenosis. The writer cannot too strongly condemn, however, the custom of giving digitalis as soon as the lesion of mitral stenosis is discovered, and while there are practically no symptoms, and there is a regular and almost too high pulse tension. But, of course, in every form of heart disease it is the evidence of *muscle failure* that alone constitutes the justification of the use of digitalis and the other drugs of its class. The pernicious habit of giving such drugs as soon as a lesion is discovered is to be further condemned, in that it probably, so to speak, "spoils the case," by habituating the heart muscle to its influence, in such a way that when the time comes for actual muscle failure and for the physiological effects of such drugs being legitimately sought, the heart muscle has been so rendered abnormal with regard to them that only an abnormal response can be forthcoming. These remarks are meant, of course, to apply chiefly to digitalis, but the same meddlesomeness may make use of other tools.

In certain of the lower animals death from digitalis poisoning is associated with tetanic contraction of the heart, which of course brings the circulation to a standstill quite as effectually as the most complete paralysis, but in the human subject all post-mortem evidence goes to show that the ventricles are paralysed and not tetanic when death occurs under the influence of digitalis. Nevertheless the fact need not shake our faith in digitalis as a muscle stimulant, inasmuch as with most active drugs the excessive action is the opposite in nature from the feeble and moderate, and "stimulation followed by paralysis" is an oft-told tale in descriptions of the effects of poisons.

When we overdo the effects of digitalis it is paralysis and not spasm of the heart that we must apprehend, but, most fortunately, in one sense digitalis is the safest of poisons inasmuch as it gives to the *observant* prolonged and emphatic warning when it is being pushed too far. This, however, is absolutely true of cases in which rest in bed is insisted upon. When digitalis is given to patients "going about," as the phrase is, it must be given in exceedingly small dose and—what is more important even—in *short courses.* That is to say, the drug must be intermitted for several days between each course, for its effects take a considerable time to pass off. There are few drugs, however, that in their administration require so much carefulness on the part of the practitioner as digitalis. It is necessary, therefore, that we should consider the mode of application of the remedy in some detail, for misused it becomes a dangerous poison—peculiarly dangerous in the fact that the symptoms of poisoning may readily be *overlooked until the sudden lethal termination of the case*, and in that the patient may be *poisoned without ever having taken a single dose beyond that rightly laid down by authority as safe.* The drug is "cumulative," as it is termed; its toxic effects are developed by *prolongation* of administration in *small and infrequent doses*, without the intervention of a pause sufficiently long to permit of the elimination of its influence. In many cases, fortunately, vomiting and abdominal pain give warning of over-action, while the pulse usually becomes slow and often bigeminal or irregular. But the careful practitioner will seldom let the administration proceed so far as the production of this condition, for he will anticipate and obviate its occurrence. All the official preparations of digitalis are reliable enough, and it is hard to choose between them. The tincture is probably most frequently used, but the infusion seems to be the most active preparation. The powdered leaves are admirable for a method of administration, similar to that to be immediately recommended with regard to a French preparation that the writer has used for many years almost to the exclusion of the official preparations. He has, however, employed this preparation simply for the sake of convenience, and not because he believes that it possesses any inherent virtue

absent in the ordinary preparations. Of the activities of the crude drug it may be said to possess both the best and the worst—the most potent and most cumulative. This preparation is the granule of M. Nativelle of Paris, containing $\frac{1}{240}$ of a grain (or $\frac{1}{4}$ of a milligramme) of "chemically pure crystallised digitaline." Into the question of the precise nature of this active principle the writer will not enter, but with regard to its activity and its equal distribution in the granules he has had large experience. M. Nativelle makes also a syrup of digitaline, each teaspoonful of which is equivalent to a granule, and contains $\frac{1}{240}$ grain of "crystallised digitaline." This is a convenient preparation for use, when a smaller dose than ordinary is required, as in the case of children.

When the writer first began using the granules of Nativelle, he believed the statement that one granule was equivalent to ten minims of tincture of digitalis, B.P., and therefore that a granule may be administered precisely in the same way as he then was in the habit of administering the tincture, that is to say, three times a "day," equivalent to three doses in twelve hours instead of twenty-four hours, or, one may say, every four hours during the day proper, and none at all during the "night," or remaining twelve hours. The result was almost invariably toxic symptoms, so that for a considerable time he gave up the use of the granules altogether. Evidently the granule consisting of one ingredient of the crude drug and the official preparations of the latter should be regarded as incomparable, and even of the latter it is a faulty mode of administration to give three doses in the first twelve hours and none at all in the second twelve hours. When it is desired to "push" quickly the action of digitalis, three granules may be given in twenty-four hours, but each must be given after an interval of *eight*—not four—hours. Usually, however, one granule in twelve hours is amply sufficient—two a day. Even this number can seldom be continued long with advantage, and usually in a very few days one granule in twenty-four hours suffices. In a few days more the drug may be stopped altogether, or intermitted for forty-eight or seventy-two hours according to circumstances. The dose must never be increased after the administration has continued for some time, a rule the necessity of which is obvious from the cumulative property of the remedy, and the dose must always be diminished as the course proceeds for the same reason.

The quantity of urine passed by the patient forms by far the best guide to the effects of digitalis, and with reference to it we must bear in mind that digitalis is a slow diuretic, requiring at least forty-eight or seventy-two hours for the production of polyuria. Thus if a patient, on the day after the one on which digitalis was first administered, gets a profuse diuresis, such diuresis cannot be attributed to the drug; all experience is opposed to such a diuresis being the effect of the drug, in all probability it is "spontaneous" or the effect of rest. But if the diuretic effect of digitalis is never obtained promptly, it is occasionally delayed for a considerable number of days. A curious experience is the following: a patient has been taking digitalis, say for ten days, while there has been no material increase of the urine-flow, and the practitioner, despairing of success, stops the remedy, and orders a drug without diuretic property, say liq. strychniæ, when, to his surprise, within seventy-two hours after the last dose of digitalis a profuse diuresis sets in. Such an occurrence is by no means rare, and indicates that the administration of the drug has been pushed too far, its effects being possibly exerted specially on the arterioles, which become too much contracted in the kidneys to permit of a diuresis which is rendered possible only when the vessels have relaxed to some extent under the diminishing influence of the drug. The possible advantage of combining a vaso-dilator with digitalis will be referred to later. When a diuresis is thoroughly established it is well to diminish the dose of digitalis, or to give the same dose less frequently, seeing that its effects thereby will be efficiently maintained, while the risk of them being over-exerted is obviated. The most difficult question is, what to do with regard to continuing the administration in cases in which there has been no response by diuresis at all. If in doubt, the rule should be to intermit the drug for at least a couple of days and probably longer. The desirableness of resuming the drug or replacing it will then have to be considered afresh.

Strophanthus and other Cardiac Remedies

The drug that undoubtedly ranks next after digitalis in the treatment of heart disease is strophanthus, usually prescribed in the form of tincture. From experimental results it would seem to have an advantage over digitalis in some respects, exerting less influence on the peripheral circulation in the direction of contraction of the arterioles. It is not quite clear, however, that this is an unqualified gain; moreover, we have found that the results of the experimentalist in his laboratory and of the physician in his ward are not entirely in accord with regard to such a decisive question as that having reference to the ultimate effects of digitalis upon the heart: in fact as to whether the heart under the full influence of this drug is found after death in systole or diastole. Of one thing the writer is convinced from his own clinical observations, and it is this: that in the case of patients who are admitted to hospital with low arterial tension, and who ultimately recover a high degree of arterial tension, the diuresis that is the indication of the successful administration

of digitalis precedes in the great majority of cases the recovery of arterial tension.

Strophanthus seems to be certainly a less cumulative drug than digitalis, but, if its administration be long continued, similar toxic symptoms, including abdominal pain, vomiting, and irregular action of the heart, are apt to be developed. The writer possesses charts to show that sometimes strophanthus will succeed after digitalis has apparently failed to produce a diuresis, and *vice versa.* In the former case, however, it is at least open to question if the influence of the digitalis has not had a share in the result. He has not found strophanthus useful in irregular action of the heart *per se*; he has thought, indeed, that it at times aggravated the irregularity. He has, moreover, been quite unable to formulate rules for guidance in the preference of strophanthus and digitalis, and he recalls one case of grave damage to the mitral valves and much dilatation of the heart in a rheumatic girl, in which digitalis always failed to benefit in the slightest degree, while strophanthus as regularly was followed by the best results, the experiment being repeated on several —many—occasions during the long period of the patient's residence in hospital. It may be added that the pulse tension was very low, altogether in opposition to theoretical considerations concerning indications for the preference of strophanthus.

There are numerous drugs capable of producing diuresis in heart disease, which are seldom given a fair chance, seeing that they are only used after digitalis at least has failed, and often after both digitalis and strophanthus have done so: convallaria, apocynum, scoparius, etc.

Then there is the series of drugs that seem to act on the kidney, rather than the heart, in the production of diuresis—diuretin (Knoll) is probably the best example of this class. The writer recalls an exceedingly grave case of aortic incompetence which seemed steadily going from bad to worse in spite of digitalis, strophanthus, etc., but in which there was the remarkable (under the circumstances) fact of the absence of even a trace of albumin from the urine. Basing his action on the soundness of the kidneys, he ordered diuretin—2 grs. every hour—with the result of establishing a profuse and long-maintained diuresis, which removed all dropsy and enabled the patient, to the marvel of physicians and nurses alike, to leave the hospital for his own home, much relieved. In cases neither cardiac nor renal, again, he has found the drug useful in setting up a diuresis for the purpose of removing local dropsies of inflammatory origin, as pleuritic effusion. Sometimes the drug produces sickness, which prevents the continuance of its use. It is the diuretic of all others whose effects the writer has been able repeatedly to reproduce at intervals. It is probable that diuretin acts specially by stimulating the kidney, and the writer believes that he has seen it twice produce hæmaturia in cases of tubular nephritis, as if by over-stimulation. Citrate of caffeine, to which diuretin is closely related, is another drug that acts principally on the kidney; most admirable results are sometimes obtained by its use, but it is somewhat capricious—or perhaps seemingly capricious—in its effects, of which we have not yet a perfect knowledge.

In considering the records of the past concerning the treatment of heart disease, one cannot fail to be struck by the high place among remedies given by the old physicians to purgatives. That these were often used to the detriment of the patient cannot be doubted, but it is no less certain that our forefathers, who were keen observers, based their practice on a measure of experience. At the present time it is *discrimination* in the use of purgatives that is the desideratum. Such discrimination must be based chiefly upon the condition of the pulse, or, to put the matter more precisely, upon the arterial tension. It cannot be too strongly insisted upon, that the finger must not be trusted to indicate the degree of vascular tension with absolute precision, while a sphygmogram can be taken easily within five minutes even in difficult cases, and at once indicates the nature of the pulse with which we have to deal. It is necessary here again to refer to the fact, already previously emphasised, that a high degree of tension is not rarely—not possibly— but is *commonly* associated with a labouring failing heart. No doubt, provided the cardiac failure be progressive, so will be progressive the lowering of arterial tension, but the process may be a long one, and of immense importance is the fact that the patient may not survive it.

That a sharp purgative by its action tends to reduce arterial tension is certain, and we cannot be surprised that it is so, in view of the tremendous influence exerted by the state of the vaso-motor nervous mechanism of the abdominal vascular system upon the distribution of the blood as a whole, as we see that influence exerted after surgical shock. The mere contemplation of such an influence may well give us pause, when we think of ordering a sharp purge in an advanced cardiac case with the pulse flickering at the wrist. Few medical practitioners of large experience can look back over many years without recalling the disastrous effects of a purgative given when the heart was very weak, and it need not be a "diseased heart" in the ordinary acceptation of the term. Even the use of an enema under like circumstances is well known to be rarely followed by disastrous—even lethal —consequences. These remarks must suffice to warn the reader against the indiscriminate use of sharp purgatives. On the other hand, in cases of high arterial tension with labouring heart, they bring much aid to the overburdened organ by lightening its load, and no doubt help to clear the blood of injurious toxins.

Space will not permit our considering the *classes* of purgatives *seriatim*, and we will refer only to special drugs and compounds. Few purgatives can rival the pulv. Jalapæ co. of the B.P., which, however, at least in hospital practice, has to be given in larger doses than the official—60 grains being not at all too much in many cases. Sir William Broadbent believes that mercury has special virtue in lowering arterial tension, and in this relation its well-known antiseptic powers in relation to toxins in the alimentary canal are worthy of note. On theoretical grounds the saline purgatives may be thought to be specially applicable in the treatment of heart disease, as the free exudation of fluid they occasion into the intestine must relieve the congestion of the portal circulation and of the liver. For this purpose they ought not to be administered largely diluted. But when given alone they do not stimulate the peristaltic movements of the bowel sufficiently to ensure the quick removal of the exuded fluid, so that it is actually possible that this may be later reabsorbed into the vessels. It is desirable, therefore, to combine the salines, of which sulphate of magnesium is the one most commonly used in the treatment of heart disease, with some drug capable of stimulating the bowel to increased peristalsis. The "black draught" of most hospitals is some such combination, to which a carminative is added. One sharp purge is better than the frequent repetition of less active ones; we hold that patients with heart disease should not be exhausted and their hearts thus still further debilitated by frequent or continuous purgation. The great indication for purgation is high arterial tension, and the many forms of cardiac failure so associated no doubt formed the ground on which the common practice of purgation in heart disease was based, that is to say, it is in cases of this kind that the best results are obtained from purgatives.

There is one remedy that so strongly makes for righteousness in treatment that it cannot possibly be ignored in an account of the management of cardiac cases:—It is morphine administered hypodermically, and usually guarded by a minute dose of atropine. Nevertheless, its use is directed to the relief of one distressing symptom only of cardiac failure, and not to the restoration of the circulation. The symptom in question is *dyspnœa*—the symptom which surpasses even angina pectoris as a cause of suffering when we regard its frequency and duration in comparison with those of angina. To Dr. Clifford Allbutt we are indebted for the introduction of this precious remedy. It should always be given hypodermically, and, as a rule, combined with atropine. It is impossible to lay down rules in detail as to dose. The treatment should be begun with infinitesimally small doses in order to see how the patient bears it, and then it is easy to increase the dose up to the point of affording relief without incurring risk. The frequency of albuminuria in cardiac cases without serious implication of the kidneys must be borne in mind. Moreover, in the distressing dyspnœa of the cardiac failure of actual Bright's disease the writer has repeatedly found the remedy to act like a charm, and to prolong life in comparative comfort, only in cases of the kind an exceedingly small dose, say $\frac{1}{20}$ of a grain well guarded with atropine, must be used at first, though it will often be found that $\frac{1}{4}$ of a grain of morphine can be given with perfect safety and with perfect effectiveness. Blocking of the bronchial tubes with secretion the writer regards as a stronger contra-indication as to the use of morphine than kidney complication. In heart cases which require no remedies directed immediately to the heart, but in which it is all-important to maintain the general health at a high standard, a mixture of liquor strychniæ and liq. arsenici hydrochlor. 4 or 5 minims of each, given in water simply and after meals, will be found very useful. Occasionally it is well to stop the arsenic, and give the strychnia alone, and before instead of after meals. Iron, no doubt, is an excellent general tonic, and a precious remedy when there is anæmia in a heart case; but the writer is inclined to think it has become too much a matter of routine to give it to patients suffering from heart disease, and in most of those in middle life and older, whose cardiac failure is apt to be associated with high arterial tension, he thinks it often does harm rather than good.

Sleeplessness is not rarely a distressing symptom in heart disease, and one which in it, as in pneumonia and typhus, taxes the resource of the physician to the utmost. Timidity and rashness must be equally eschewed. Those who err on the side of the former the writer would remind that sleeplessness is a terrible "tendency to death" in all these diseases, and that to let a patient die without attempting to save him is as bad as running risk in the attempt. But happily, with ordinary care, such risk is infinitesimal, and lies specially in lung and kidney complications. In heart cases insomnia is often associated with dyspnœa, and the remedy which is most efficient for the latter is best for the former, namely, morphia, given hypodermically. Among other drugs that may be used for sleeplessness are chloralamide and urethane. The approximate dose of the latter—which must be determined by the circumstances of the case—should be kept dissolved in sp. vin. rectif., and added to water at the time of administration. With regard to urethane, it is a safe but a weak drug, and has generally to be given in large doses. Shortly after the drug was introduced, a patient of the writer's, a bad rheumatic heart case, received by accident an overdose, but the only undue consequence was that the patient, as well as sleeping during the night, slept nearly

all the following day, respiration and circulation being well maintained, and only benefit resulting.

There are very few heart cases, other than those associated with aortic disease in the wide sense, that depend on muscle failure promoted by or actually dependent on syphilitic lesions of the myocardium,[1] and the possibility of anti-syphilitic therapeutics being beneficial, if not curative, of the case must not be lost sight of. The patient will probably have indications of heart failure, the common antecedents of which are absent while he has had syphilis. It must be remembered, however, that because a lesion is syphilitic it is not therefore removable by mercury and iodide of potassium; the tissue destroyed cannot be restored, although the neoplasm may be atrophied.

A drug that has come greatly into vogue of late is liquor strychniæ (or an equivalent preparation) in the treatment of advanced heart disease. The writer regards it as essentially a respiratory stimulant, and of its value as such in these cardiac cases there can be no question. It is best given hypodermically, but this method is naturally not to be recommended until the case has become very serious. Atropine has a somewhat similar stimulant action on the respiratory centre, but its use is almost confined in the treatment of heart disease to guarding, as it were, the use of morphine. Strychnia may be used for the same purpose.

The use of strychnia in early stages of heart disease as a general tonic has been already referred to.

The consideration of the treatment of the special ailments that result from the disturbed circulation of the various organs in heart disease may be carried to indefinite length, but too much attention to these is apt to throw into the background the great and essential "tendency to death," namely, *heart failure*, of which these ailments are for the most part comparatively insignificant consequences. If we succeed in restoring the heart, all such ailments will speedily be relieved. On the other hand, the most efficient treatment directed to local consequences in the different organs must fail, if no improvement in the general circulation is accomplished, but this statement must not be taken to imply that local symptoms are not to receive local treatment according to the dictates of common sense and common medical knowledge.

Heart Block.

By this term is meant an interruption in the wave of contraction, which, passing from the auricles to the ventricles, constitutes the normal beat of the heart. When the wave is blocked, instead of the auricular and ventricular contractions occurring in harmoniously co-ordinated sequence, they become dissociated and independent of one another. The ventricular contractions begin to take place much less frequently than those of the auricles, and as a result bradycardia is established. This bradycardia, when, as is often the case, associated with syncopal or epileptiform attacks, is known as Stokes-Adams disease. It is now certain that this complex of symptoms is always due to heart block, and has for its pathological basis a lesion of the conducting fibres between auricle and ventricle.

With the elucidation of the anatomical, physiological, and clinical aspects of heart block we must associate the names of Kent, His, Gaskell, Wenckebach, Erlanger, Hering, Mackenzie, Gibson, and Tawara, all of whom have made noteworthy contributions to the subject.

Anatomical and Physiological.—The wave of contraction normally passes from auricles to ventricles by way of the auriculo-ventricular bundle of Kent and His. The fibres of this bundle differ structurally from the rest of the heart muscle, and their course can therefore be traced (as has been done by Tawara) by special methods. They form a system ramifying under the endocardium, and serving to co-ordinate the movements of the chambers of the heart. Broadly, they are distributed in this fashion. The central point in the system is a knot, or plexus of fibres, lying above the attachment of the middle cusp of the tricuspid valve. From this nodal point the fibres run down in two main bundles, one to each ventricle, which subdivide, forming the trabeculæ, and end in the papillary muscles. They are insulated from the rest of the heart by a connective-tissue layer. The left main trunk can often be seen in elderly persons as a whitish band measuring one-fourth of an inch across below the pons membranaceæ septi. The nodal point is brought into connection with the auricles by the auriculo-ventricular bundle, which extends from the coronary vein along the right side of the inter-auricular septum to the position of the knot. The auricular fibres of the system are finer than the ventricular, and like them ramify in the walls of the chambers from which they arise.

By experiment upon animals it has been ascertained that the time required for the transmission of an impulse from auricles to ventricles along the *a.-v.* bundle is $\frac{1}{5}$ second. The impulse can be checked more or less completely by compressing the bundle more or less forcibly. When this is done an interesting series of phenomena follow. Slight pressure causes a progressive lengthening of the periods which elapse between successive auricular and ventricular contractions, until at length one ventricular beat is dropped

[1] *See also* "Syphilis."

—the chambers failing to respond to the auricular stimulus. When this takes place the succeeding auriculo-ventricular period is unusually short, owing to the brief rest of the *a.-v.* bundle having improved its conductivity. As the compression of the *a.-v.* bundle becomes greater, the omission of ventricular beats occurs more frequently; thus at first there may be failure on the part of the ventricle to respond to every eighth auricular contraction (8 : 1 rhythm), while with further compression a 2 : 1 rhythm is reached, in which only every alternate auricular contraction is followed by ventricular activity. With greater compression rhythms in which only every third or fourth auricular wave is followed by a ventricular beat (1 in 3, 1 in 4, . . . rhythms) occur. When a 1 in 3 rhythm is established, however, complete heart block usually occurs, the ventricles asserting their independent rhythm, and becoming completely dissociated from the auricles. When in place of producing heart block by gradual compression, the *a.-v.* bundle is suddenly lightly clamped, the ventricles stop, but the auricles remain unaffected. The pause in the action of the ventricles endures a minute or more, and then the independent, slow, ventricular rhythm develops. When partial block is transformed into complete block the same momentary check in the ventricular beat occurs. All the above phenomena have a very important bearing on heart block in man, which shows a striking analogy to that produced experimentally. When heart block is established, stimulation of the accelerator nerve affects both sets of chambers, but stimulation of the vagus, or its paralysis by atropine, affects the auricular rhythm only.

Clinically, heart block reveals itself by a persistently infrequent pulse — 30, or less per minute. It is as a rule a disease of the later decades. To establish the diagnosis of heart block, simultaneous tracings must be taken of the movements of the vessels of the neck and of the radial artery. To describe such tracings in detail would unduly burden this article; for further information reference should be made to the writings of Gibson and Jas. Mackenzie, to the latter of whom, in particular, we owe so much of our knowledge of the employment of this means of diagnosis. It is enough to say that by comparing the two sets of tracings the relation of the auricular to the ventricular beat can be determined. Normally, in the tracing obtained from the neck the interval of time which elapses between the oscillation produced by the auricular contraction and that produced by the carotid pulse (*a.-v.* interval) is $\frac{1}{5}$ second. Prolongation of this implies a retardation of conductivity in the *a.-v.* bundle. A further stage of block is shown by the occasional omission of a ventricular beat, followed, as in experimental block, by a diminution of the succeeding *a.-v.* interval on account of increased excitability following the brief rest. Finally, it may be that complete block is present, the infrequent carotid pulsations having ceased to bear any relation to the auricular waves. Dissociation of the auricular and ventricular movements may also be demonstrated on the fluorescent screen, by which the independent contractions of the two sets of chambers are made visible to the eye, and also, as Gibson has shown, by observing the oscillations of an electrometer connected with the apex beat and base of the præcordia. As in animals, atropine increases the rate of the auricles, but does not affect the ventricles.

Many of the clinical features of bradycardia and Stokes-Adams disease have been considered elsewhere, and need only a brief mention here. The bradycardia is usually permanent. The characteristic seizures vary in frequency and severity. They may consist merely of a dazed feeling of short duration, with some restlessness and faintness; from this, up to serious fatal syncope attended by convulsions and Cheyne-Stokes respiration, all grades occur. They have the common feature, however, of being attended by a cessation of the pulse lasting for several seconds, and when the beat returns it may for some time be less frequent than prior to the seizure. Whether this temporary abrogation of the action of the ventricle was the cause or the effect of the seizures, was for long in dispute; it is now, however, agreed that in these cases the stoppage of the pulse precedes, and probably is the cause of the attacks. Syncopal attacks may supervene either in partial or complete block, and perhaps also when the heart is acting normally. As we have seen, experimental complete block, whether sudden or gradual, is attended by a temporary cessation of the ventricular beat, and it is reasonable to suppose that clinically the same thing takes place. When the conductivity of the *a.-v.* fibres is lowered by disease, any alteration of the auricular rhythm, such as may happen reflexly through vagus action, may be enough to initiate the independent ventricular rhythm. The hypothesis that syncopal attacks are due to the transformation, from any cause, of partial into complete block, is inadequate to explain their occurrence in cases which already present the phenomena of total block. It may be, as Hay and Schmoll suggest, that in these cases there is some direct interference with the operation of the intra-ventricular stimuli—*i.e.* the stimuli which arise in the ventricle itself, and regulate its independent contraction.

Morbid Anatomy.—A sufficient number of examinations of hearts of patients suffering from heart block have now been made to warrant the assertion that the essential change is a lesion of the auriculo-ventricular bundle. The lesion may be of the nature of a gumma, or an invasion by scar tissue, with cellular

infiltration, and loss of striation of the fibres. The vagus and heart muscle have been found healthy.

Heartburn. *See* Indigestion (*Symptoms, Sensory, Cardialgia*); Pregnancy, Affections and Complications (*Digestive*); Stomach and Duodenum, Diseases of (*General Symptomatology, Heartburn*).

Heart, Congenital Malformations of.

In commencing the subject of congenital malformations of the heart we cannot do better than begin with a classification of its various forms taken from Professor Vicrordt's admirable monograph. This list gives some idea of the complexity of the subject and of the extraordinary multiplicity of forms which congenital defect of the heart may assume. In estimating this we must further bear in mind that it is much commoner to find several malformations combined in one heart than to meet with one only, and that in the case of patients who have attained adult age there are apt to be further complications as the result of post-natal endocarditis having attacked the abnormal parts.

Vierordt's classification is as follows:—

1. Patent foramen ovale, uncomplicated defects of the inter-auricular septum.

2. Uncomplicated defects of the inter-ventricular septum.

3. (*a*) Primary inflammatory stenosis of the ostium and conus of the pulmonary artery without malformation (with patent foramen ovale).

(*b*) Stenosis and atresia of the pulmonary artery.

(1) Without septal defect and transposition of the vessels.

(2) With septal defect and transposition of the vessels.

4. Transposition of the large arterial trunks (with various complications).

5. Dilatation of the pulmonary artery.

6. Persistence of the truncus arteriosus (including cases of direct communication between the aorta and pulmonary artery).

7. Congenital stenosis and atresia of the commencement of the aorta (conus and ostium).

(*a*) From arrest of development.

(*b*) From fœtal endocarditis.

8. Congenital abnormalities of the semilunar valves.

9. Stenosis of the aorta near the entrance of the ductus arteriosus, persistent isthmus aortæ.

10. Congenital narrowness of the aortic system.

11. Patency (uncomplicated) of the ductus arteriosus.

12. Primary defects at the right auriculoventricular opening—

(*a*) Developmental.

(*b*) From fœtal endocarditis.

13. Primary defects of the left auriculoventricular opening—

(*a*) Developmental.

(*b*) From fœtal endocarditis.

14. Misplacements of the heart.

15. Malformation of the pericardium.

Causation.—The lesions of congenital heart disease may be divided into three groups according to their causation.

(1) In some cases the abnormalities in the heart are evidently of such a nature that they must have resulted from intra-uterine endocarditis. When this disease occurs in fœtal life it may be of the warty variety, especially when it affects the semilunar valves of the arterial openings. In the great majority of cases, however, it assumes the sclerotic form, and leaves behind it an opaque white or buff yellow thickening of the endocardium. It may cause contraction of one or more of the cavities or of the valvular openings, or shrinking or adhesion of the valves themselves. The condition occurs more frequently on the right than on the left side of the heart.

(2) In many cases, part or the whole of the lesion consists in persistence of openings in the inter-ventricular or inter-auricular septa or of the ductus arteriosus. Abnormal patency of any of these structures is referable to obstruction to the course of the circulation occurring at a period earlier than that of their natural closure. This obstruction may exist in the lungs or in the course of the pulmonary artery, in the right ventricle or at the tricuspid aperture; and it may equally well be situated on the left side of the heart either at the mitral or aortic orifice. The presence of a patent inter-ventricular opening indicates an earlier date for the commencement of this obstruction than an open foramen ovale or ductus arteriosus only. Sometimes on examining the heart and lungs no lesion can be found which could have caused any obstruction to the circulation; in such cases it is probable that the block has been of a temporary nature and has disappeared.

(3) The remainder of the cases, which cannot be grouped in one or other of these categories, present other imperfections of development which show no trace of being due to inflammatory action. Such are transposition of the large arterial trunks, congenital abnormalities of the semilunar valves, absence of the pericardium, etc. The causation of these arrests or perversions of development is in

many cases exceedingly obscure. The experiments of Geoffrey St. Hilaire and other more recent observers have shown that a variety of mechanical and chemical influences (such as violent shaking and the injection of chemical irritants or of pathogenic germs and their toxins) acting on an egg often result in congenital malformations in the chicken. The causes which under ordinary circumstances produce a like result in the human embryo are as yet quite unknown. It is extremely probable, however, as Ballantyne has pointed out, that the same morbific influences which occasion endocarditis in the later stages of intra-uterine life give rise to developmental defects when they act during the earlier months. In other words, the morbid influence which in the young embryo produces a teratological result has a pathological effect when it acts on a fœtus which has so far developed as to have differentiated organs.

It is important, in connection with the causation of congenital heart disease, to draw attention to the fact that a great variety of external malformations due to imperfect development (such as hare-lip, imperforate anus, webbed fingers, etc.) are frequently found along with it, and that it has been pointed out recently that a curiously large proportion of "Mongolian" imbecile children present congenital heart lesions. This association with the other defects of development seems quite as marked in instances of fœtal endocarditis as in the so-called developmental cases.

Clinical Phenomena. — The symptoms met with in cases of congenital heart disease vary greatly according to the extent as well as the nature of the lesions present, and the degree to which these interfere with the circulation. In some few cases (*e.g.* in some of septal defect) there may be no symptom or physical sign that could lead to a suspicion of abnormality during life. In others (*e.g.* in many of patent ductus arteriosus) there may be nothing discoverable beyond a murmur. In the great majority of cases, however, we meet not only with murmurs and other abnormalities on physical examination of the heart, but also with more or less deep cyanosis, with chilliness of the extremities and concentration of the blood; and we also often find clubbing of the finger-ends. If the interference with the circulation is marked we find some considerable degree of debility, and there may also be a varying amount of dyspnœa, attacks of cardiac pain, epistaxis, and epileptiform seizures of various types. The coincident occurrence of external defects of development has been already alluded to.

Some of these clinical phenomena deserve further mention.

Physical Signs.—The physical signs of congenital heart disease discoverable on palpation, percussion, and auscultation consist of alterations in the position or force of the heart's apex-beat, thrills, increased size or altered contour of the cardiac dulness, murmurs, and changes in the loudness of the sounds—especially of the pulmonary second sound. The combination of these phenomena met with has often much more significance than their individual occurrence. Thus a loud systolic murmur, with no increased dulness and no accentuation of the pulmonary second sound, has a very different meaning from an otherwise identical murmur accompanied by the usual signs of cardiac hypertrophy.

The murmurs are usually peculiar in their areas of audition, and in their lines of propagation, which do not correspond to those characteristic of any valvular lesion. It is often difficult, if not impossible, to determine the area of their maximum intensity, and this may also vary from time to time in the same case. In the very great majority of instances the murmurs are systolic in time; rarely, however, they may be diastolic or presystolic. Peculiar humming sounds are sometimes met with, especially over the base of the heart. It is to be remembered that murmurs due to congenital cardiac defects may change considerably in character and distribution if the patient becomes anæmic.

Cyanosis.—Cyanosis is present in the majority of cases of congenital heart disease, and it is so characteristic of these cases that they have been spoken of as instances of "morbus cœruleus." The discoloration varies greatly in degree. When well marked it is deeper than that produced by almost any other pathological condition. It is visible all over the body, but is especially noticeable in the extremities (fingers, nose, ears, etc.), and on the visible mucous membranes. When severe it may be accompanied by some puffiness of the features, but œdema of the extremities is rare, and only occurs late in the course of the case. The cyanosis is usually present at birth and persists during life, but it may only come on when the patient is several years old. Its degree varies from day to day according to the general health of the patient.

The cyanosis of congenital heart disease differs from other forms of cyanosis in degree rather than in kind. It is important to remember, however, that cyanosis is not met with to any appreciable extent in childhood as a result of post-natal endocarditis; its presence always, therefore, indicates a congenital lesion (either developmental or from endocarditis).

The causation of the extreme cyanosis in these cases has long engaged the attention of the medical profession, and several explanations of it have been suggested.

(1) The view that it is due to admixture of the venous and arterial blood allowed by their defective separation in the abnormal heart is to

be considered no longer tenable. Extreme cyanosis has been found in cases where the condition of the heart did not permit of any abnormal mixture of this kind; and, again, individuals have been known to live for years without a trace of cyanosis, although their hearts presented more or less extensive septal defects.

(2) A more widely accepted explanation is that first enunciated by Morgagni, that the discoloration is merely the result of congestion of the venous system arising from backward pressure. It seems probable that this accounts to some extent for the phenomenon, and the differences noticed between the cyanosis of ordinary heart and lung cases and that of congenital malformation may be partly at least attributable to the very early onset of the backward pressure having caused greater dilatation of the smaller blood-vessels. The fact that the blood which distends these is abnormally dark from its concentration must also be borne in mind.

(3) Dr. Lees has presented strong arguments in favour of the cyanosis in congenital heart cases being simply an index of the extent to which aeration of the blood in the lungs has been hindered, the discoloration being due, not to venous congestion pure and simple, but to the congestion of non-aerated blood.

Clubbing of the Fingers and Toes.—This is a common symptom of congenital heart disease; but the exact conditions under which it occurs are not quite clear. It is present in most cases where the cyanosis is extreme, but not in all, and it may be present without any cyanosis. Dr. Lees points out that it is not likely to occur if the systemic venous congestion is prevented by the presence of a widely patent foramen ovale. When clubbing of the fingers and toes is present to a marked degree a corresponding condition of the nose and ears is usually seen.

Blood.—In cases of this form of cyanosis, as well as in others, there is a concentrated condition of the blood. Its specific gravity is increased (1070-1080), and the number of coloured blood corpuscles may reach as high as from 8,000,000 to 9,000,000 in the cub. cm., while the hæmoglobin may be over 150 per cent.

Some Forms of Cardiac Malformation.—A few facts may now be given regarding some of the varieties of congenital heart lesion, including those which are most frequently met with in clinical work or in the post-mortem room.

Patent Foramen Ovale.—Very little blood passes through the foramen ovale at birth, although the opening is often not completely closed before the middle of the first year. Not uncommonly the foramen persists during life in the form of a small valvular opening. This scarcely amounts to an abnormality, and gives rise to no symptoms. When, however, the opening is large and not valvular, it is to be regarded as distinctly abnormal, even if, as sometimes happens, it has no apparent effect on the health. In some cases the patent foramen is accompanied by a more or less extensive defect of the adjacent inter-auricular septum. Patency of the foramen ovale is not very rarely the only malformation discoverable; it is, however, much more common to find it along with other malformations of the heart or large vessels.

The symptomatology of this defect is still very obscure. Frequently it gives rise to no symptoms or physical signs at all. Such symptoms as have been ascribed to it are inconstant and ill-defined. Murmurs of various kinds have been met with, and these have not only been presystolic or diastolic, as might have been expected, but quite as often systolic in time. The area of their maximum intensity has also varied, but they have generally been heard best about the level of the 3rd or 4th costal cartilages. In some cases there has been cyanosis; in others it has been absent. Under ordinary circumstances the diagnosis of these cases is impossible. In the rare instances, however, in which a patient with this defect acquires mitral incompetence in later life, the defective state of his inter-auricular septum may be betrayed by the occurrence of venous pulsation in the neck without evidence of tricuspid disease.

The prognosis in these cases is obscure. Many of the patients die in early infancy, but not a few instances are on record of individuals with large defects of the inter-auricular septum who have reached adult life and even old age (sixty to eighty years). It must be remembered, however, that the presence of an open foramen ovale constitutes a source of danger to life in cases in which anything of the nature of venous thrombosis is present. A detached fragment of a thrombus carried to the right side of the heart is very apt to pass directly into the left auricle, thence through the left ventricle into the arterial system, causing an embolism in the brain or elsewhere.

Defects in the Inter-ventricular Septum.—The normal inter-ventricular opening closes by the eighth week of intra-uterine life. A permanent opening in this situation constitutes one of the commonest malformations of the heart. It may occupy the position of the "undefended space," or membranous portion of the septum, or it may be at one or other side of this area. It is but rarely situated near the apex. Although occasionally found alone, this malformation is generally one of several. It is especially apt to be associated with pulmonary stenosis. When it is met with to a very marked degree along with a large defect of the inter-auricular septum, we have the condition which has been described as "corbiloculare" or "reptilian heart."

The physical signs produced by defects in the inter-ventricular septum are a matter of great difference of opinion. It is generally believed, however, to produce a loud, harsh, long-continued systolic murmur, which is audible chiefly over the upper third of the præcordia, in front, and also markedly in the inter-scapular region behind, and which is not accompanied by any thrill. Many differences in the physical signs have, however, been described by good observers. Cyanosis may be absent; and there is no question that very large defects in the inter-ventricular septum may persist without causing any symptom or physical sign whatever.

The prognosis is, on the whole, less favourable than in cases of open foramen ovale. The presence of an inter-ventricular communication causes greater interference with the circulation, and leads at an earlier period to cardiac hypertrophy. Cases have, however, been recorded in which patients with this lesion lived forty and even forty-five years. The danger of the occurrence of embolism is the same as in cases of patent foramen ovale.

Stenosis and Atresia of the Pulmonary Artery.—This is probably the commonest congenital malformation of the heart.[1] It may be due either to abnormal division of the common truncus arteriosus in the course of development, or to the result of fœtal inflammation. The exact situation of the narrowing varies. It may be caused by thickened and adherent valves, or may be situated above the valves. The artery itself may be thickened, or there may be a diminution in size of the conus. The lumen of the vessel may be quite obliterated. It is usually accompanied by patency of the inter-ventricular or inter-auricular openings, or both, and often by an open ductus arteriosus.

The symptoms are generally striking. There is usually marked cyanosis, with its accompaniments. Often the right side of the heart shows distinct evidence of enlargement. The auscultatory signs vary considerably. A loud systolic murmur in the second and third left spaces near the sternum, which is propagated into the vessels of the neck, is what is most characteristic. There may, however, be no murmur at all. In cases of simple stenosis the pulmonary second sound is faint or absent, but in some cases, where the valves are not implicated and the ductus is widely open, it may even be accentuated.

The prognosis in cases of pulmonary stenosis is not good as regards the prospect of long life. In exceptional instances patients have been known to live more than forty years. The great majority, however, die in childhood or as young adults. In cases of atresia the chances of life are considerably less than in those of stenosis. There would seem to be a special tendency for patients with this malformation to suffer from various forms of cerebral disease. It is said, also, that they are peculiarly liable to tuberculosis of the lungs.

[1] According to Peacock, more than four-fifths of the children with congenital heart disease who reach twelve years old suffer from this lesion.

Transposition or Malposition of the Aorta and Pulmonary Artery.—This is a group of not very common cases which are complicated both as to their anatomical details and their mode of production.

The physical signs are also very obscure and equivocal. There is generally marked cyanosis, and systolic murmurs are usually present. These, however, may possibly be referable to the complications which are present. According to Hochsinger, the presence of extreme cyanosis, with pure heart sounds and accentuation of the second sound at the base of the heart, gives sufficient ground for a diagnosis of this condition in an uncomplicated form.

The prognosis in cases of transposition of the large vessels is very bad. In a few instances the patients have survived to adult age, but in the great majority they die within the first six months of extra-uterine life.

Stenosis and Atresia of the Aorta.—This is a much less common condition than narrowing of the pulmonary artery. Most forms of it are inconsistent with continued extra-uterine life, and the children survive for a few days at most. The only variety of the malformation which is compatible with longer life, and therefore of clinical interest, is the stenosis or atresia which occurs near the entrance of the ductus arteriosus. This is situated at the commencement of the descending aorta, either above or below the ductus. The obstruction at this point leads to great dilatation of the aortic behind it and of its branches, and an extensive collateral circulation develops.

The symptoms in these cases are either slight or quite absent in childhood, so that the condition is not diagnosable. They become, however, steadily more marked in later life. This is largely due to the fact that the constricted portion of the vessel remains about the same size, while the lumen of the rest of it grows with age, so that the effect of the disproportion between the two becomes increasingly felt. Cyanosis is seldom present. The physical signs which are characteristic of this malformation in adult patients are as follows:—(1) Marked hypertrophy of the left side of the heart; (2) a loud systolic murmur, accompanied by a strong thrill over the manubrium sterni to right of it, and in the jugular fossa, which is also conducted into the vessels of the neck; (3) a visible collateral circulation in the form of superficial, dilated, pulsating arteries, recognisable over the chest and abdomen, in which systolic murmurs may sometimes be heard. The diagnosis may be confirmed by the occurrence of retardation of the femoral pulse and marked weakness of the

arterial pulses all over the lower half of the body, contrasting with those of the distended vessels above.

In this form of aortic stenosis or atresia the prognosis is much more favourable than it is in cases of constriction of the pulmonary artery. In some cases its presence seems to have been consistent with a long and apparently healthy life. When death occurs it is sometimes due to rupture of the aorta, sometimes to cerebral or pulmonary lesions.

Congenital Abnormalities of the Semilunar Valves.—Abnormalities in the number and size of the semilunar valves are not very uncommon. One segment may be unusually small, or there may be two or four instead of three. Such malformations are generally of themselves of no consequence. It is found, however, that in later life the abnormal valves are specially liable to be affected by endocarditis.

Patent Ductus Arteriosus.—The ductus arteriosus rapidly closes after birth, and it should be entirely obliterated somewhere between the tenth and twentieth days of life. Failure of this normal process of involution and consequent persistent patency of the canal is a common complication of various congenital malformations of the heart and vessels, as already mentioned. Occasionally, also, cases are met with in which an open ductus is the only lesion to be found. The persistent duct may be greatly dilated, in which case it often acquires a funnel shape, the aortic being the wider end. The pulmonary artery may be greatly dilated, and the left ventricle is generally hypertrophied.

When the lesion is uncomplicated there is no cyanosis until late in the progress of the case, and the patient often lives many years in the enjoyment of good health and without any abnormal subjective sensations. Cardiac hypertrophy and other indications of some embarrassment of the circulation are, however, apt to intervene sooner or later. The murmurs vary, but that which is much most frequently heard is a loud systolic bruit in the second left intercostal space a short distance from the sternum, which is accompanied by a palpable thrill and an accentuated second sound. Occasionally there is a diastolic murmur.

The prognosis in uncomplicated cases of patent ductus arteriosus is more favourable than in most forms of congenital heart disease. About half of the published cases have survived puberty, and many have had long and active lives without any signs of disease.

Acardia.—Acardia or absence of the heart is met with in rare instances of still-born twin monstrosities.

Double Heart.—A double heart has been described in cases of extremely deformed fœtuses. Such a condition is, however, never met with clinically.

Misplacement of the Heart.—The heart may be displaced to the right side, or forwards, or even, in very rare cases, upwards into the neck or downwards into the abdominal cavity.

Dextrocardia.—Misplacement of the heart to the right may occur without any corresponding malposition of the other organs. Usually, however, it is only part of a general transposition of viscera (*situs viscerum inversus*). When it is present alone this condition causes no disturbance of the general health, but it is often associated with other malformations.

A lesser degree of misplacement to the right, so that the heart occupies a mesial position in the thorax, such as is found at a very early period of embryonic life, is occasionally met with, usually along with extensive developmental defects.

Ectopia Cordis.—Ectopia cordis or prolapse forwards of the heart is a condition which dates from a very early period of intra-uterine life. It is associated with congenital fissure or entire absence of the sternum, and sometimes with defect of some of the ribs. It varies greatly in degree, and when severe may be associated with absence of the pericardium.

Absence of the Pericardium.—Absence of the pericardium may occur along with abnormalities in the position and form of the heart or independently of other defects. It may be complete or partial.

General Diagnosis of Congenital Heart Affections.—It is not generally very difficult in childhood to determine in any given case whether a cardiac lesion is congenital or acquired. The most important points to be considered in making the diagnosis are:—The presence of cyanosis; the loudness of the murmur taken along with the age of the child; an atypical situation and propagation of the murmur, and the presence of hypertrophy of one or other side of the heart.

In children who are weakly and anæmic it is always well to be particularly cautious about the diagnosis of congenital heart lesions from the presence of murmurs alone. In such cases comparatively loud basic murmurs may be found, which disappear entirely on recovery from the general condition of debility.

The diagnosis of the exact condition is a question which must, in the large majority of cases, remain unanswered owing to its extreme difficulty. As the patient grows older the diagnosis of the lesion becomes increasingly difficult owing to the great frequency of secondary endocarditis of the congenitally abnormal structures. Hochsinger's axioms as to diagnosis, which are founded on the observation of children under five years old, but which mostly apply equally well to older patients, are (slightly abridged) as follows:—

1. Loud, harsh, musical murmurs, with a normal or but slightly increased area of dulness, are met with in little children only in congenital cases. When acquired inflammatory heart affec-

tions occur in them, with very loud murmurs, they invariably cause great increase in the cardiac dulness.

2. The occurrence of murmurs along with greatly increased cardiac dulness and feeble apex-beat in young children is in favour of congenital disease. The increased dulness depends mainly on enlargement of the right heart while the left is but slightly altered. On the other hand, acquired heart disease in children is accompanied by increased force of the apex-beat, because its effect falls first on the left side, while the dilatation of the right heart sets in later, and does not affect the increased strength of the apex-beat.

3. The complete absence of murmurs at the apex, while they are distinctly present in the region of the auricles and over the pulmonary orifice, is always an important element in the differential diagnosis, and is more in favour of septal defects or pulmonary stenosis than of endocarditis.

4. Abnormal weakness of the pulmonary second sound along with a distinct systolic murmur can only be explained, in early childhood, by assuming the presence of congenital pulmonary stenosis, and consequently is worth remembering as a point in the differential diagnosis.

5. Absence of a palpable thrill, in spite of very loud murmurs audible all over the præcordial region, occurs almost exclusively in cases of congenital septal defects, and this condition is therefore against a diagnosis of acquired heart disease.

6. Loud systolic murmurs (especially those accompanied by a thrill) which have their point of maximum intensity situated over the upper third of the sternum, and are unaccompanied by any symptom of marked hypertrophy of the left ventricle, are very important for the diagnosis of persistence of the ductus arteriosus, and cannot be explained by the assumption of endocarditis of the aortic valves.

Prognosis.—The prognosis depends on the age of the patient, the state of his strength, and the presence or absence of hypertrophy of the heart and of cyanosis. The character of the murmur is of small importance in this connection compared with the general condition. In young infants the prognosis must always be very guarded until there has been time to observe to what extent the cardiac abnormality interferes with the vital processes. If the cyanosis is marked, the child weakly, and the heart enlarging in spite of care, the outlook is very bad. If, on the other hand, the patient's nutrition and vigour have been tolerably well sustained for several years, if cyanosis and clubbing are absent or slight, and the heart little, if at all, enlarged, the chance of his reaching manhood may be regarded as fairly good.

In estimating the effect of a congenital heart lesion on the general health, it is important not to attribute to its influence debility arising from other causes. It is not very uncommon to meet with children whose general health has been neglected owing to their having a loud heart murmur, which was supposed to indicate an incurable cause for all their ill-health, but who rapidly respond to ordinary treatment of their other ailments.

Treatment.—Generally the only possible treatment consists in keeping the patient quiet and warm and attending to his nourishment. In cases where there are periodic signs of failure of compensation (increasing dyspnœa, epistaxis, epileptiform attacks, etc.), great advantage is derived from confining the patient to bed for a time and giving digitalis or strophanthus. On the habitual cyanosis of these cases cardiac tonics have no effect. For cardiac pain and breathlessness, when severe, nothing but morphine, and that sometimes only in large doses, brings any relief. If rickets, anæmia, indigestion, or any other diseases are present, they must be carefully attended to.

Heart, Neuroses of.

By a neurosis is usually meant a disorder, the manifestations of which are attributed to an influence of the nervous system, the nature of which is quite or largely unknown. Under these circumstances, signs and symptoms are apt to be raised to the rank of diseases, and words made to take the place of things.

Such a state of matters tends, naturally, to unreasoning or empiric treatment, which in the case of some so-called neuroses of the heart may be neither successful nor safe. These are divisible into two great classes representative of the two chief properties of the nervous system—sensibility and motility. They are classed as disorders of cardiac sensibility and cardiac motion respectively, and may, to all appearance, in some cases bear the relation one to another of cause and effect, as when the pain of angina is followed by a rescuing acceleration of the heart's action.

In the article dealing with the physiology of the heart (p. 66) it was pointed out that the essential factors in the functional unity of the heart's action were the muscle cell, its blood-supply, and its nervous endowment. In examining the nature and determining the preponderant character of cases when classifying cardiac neuroses, as well as in prescribing treatment, these factors have constantly to be borne in mind.

For information regarding sensory disorders of the heart the reader is referred to the article

on Angina Pectoris, and its discrimination from non-cardiac dysæsthesiæ in the region of the heart (vol. i. p. 187). In this place we shall be concerned with the nature and treatment of motor neuroses of the heart.

In the introductory chapter it was pointed out that a rhythmical pulsation of the embryonic heart was to be detected at a stage prior to the organisation of the blood-vessels and the intrusion of the nerves. Life is manifested in the cardiac muscle, in other words, before the complete formation of the vessels into which the heart is destined to project blood, and before the nerves destined to regulate its action have grown into it and been distributed to its essential cells. In course of time, however, it was also pointed out that such a distribution does take place, and is not without an influence upon its movements. Regulation of rhythmicality appears to be as necessary to its persistence as nutrition of the structures manifesting movement. The existence of rhythmical structures not endowed with nerves may now be denied as positively as the existence of such structures unnourished by blood. It is evident, therefore, that irregularities and abnormal pauses in the action of structures which are perfectly rhythmical under normal circumstances must in some measure be due, either to the abnormal exercise of the regulative influence, or to the exercise of this influence being abnormally in abeyance or impaired. It was likewise argued that the structure common to the anabolic and catabolic nerves was the muscle cell — that it was, in short, the peripheral conducting medium between the two adjuvant and supplementary if not antagonistic series of nerves. The muscular or other peripheral organic cell closes the neuro-cellular circuit at one end, just as the cells of the cortical and subcortical centres of reflex action, physical and psychical, do at the other. Continued defect, therefore, in any one of these three factors which constitute the functional unity of cardiac, as indeed of all other visceral action, may lead to disorder of the regular action normal to the organ (*see* Plate).

Regarding the second sound of the heart as a passive event, and the double contraction of the auricle and ventricle as one in propulsive effect, the normal action of the heart may be viewed as consisting of a regular alternation of action and pause, of work and rest, distributed in due and recognised proportions, the consequence of which, *ceteris paribus*, is a recognised normal *rate* of action. The latter, naturally, varies with circumstances. The heart of the human fœtus, notwithstanding its division into distinguishable chambers, is in reality a complex single tube with communicating orifices, and the systemic circulation is to all intents and purposes a rhythmically pulsating vessel propelling blood supplied to it, as a rule, from a higher level than that which it occupies itself, and always under a steady materno-placental pressure which influences it. The active and passive phases of its action are therefore practically equal. The "tic-tac" of fœtal cardiac action shows little distinction between the duration of systole and diastole. Excessive uterine pressure from interference with the oxygenation of the fœtal blood may indeed diminish the normal rapidity of the fœtal heart, but its two chief phases are of nearly if not quite the same duration. With the introduction of the second or pulmonary circulation at birth, the closure of fœtal communications, and the withdrawal of placental pressure, the increased activity of the organ requires a greater period of rest, and a noticeable distinction between the duration of systole and diastole arises, which increases with the growth of the organism, until, in adult life, the duration of diastole is distinctly greater than that of systole in the normal and unaccelerated cardiac cycle.

Under what are usually regarded as abnormal circumstances, the duration of both phases of the cycle may be equally increased or equally diminished, or one or other phase may be increased or diminished in proportion to the complementary action. Thus arise the conditions of abnormal slowness of the heart's action or bradycardia, abnormal rapidity of the heart's action or tachycardia, and irregularity in the phases of the cycle or arrhythmia. Moreover, in this arrhythmical action there may be a certain periodicity of variation which introduces a rhythmical form of arrhythmia, to use an apparently contradictory phrase, by means of which certain recognised variations are brought about, such as the bigeminal and trigeminal pulse, and the associated coupling of beats in the heart's action. Such abnormalities may be more or less persistent or even permanent, and then they become so marked a clinical feature that the apparent causes and consequences of the condition are capable of separate study as pathological entities, and are so considered in many text-books, although it is questionable whether any of them have a right to be regarded as such. Taking them *seriatim*, we shall first consider abnormal retardation of the heart's action.

Bradycardia. — Grob, who was the first to apply the term bradycardia to the condition originally described by Adams and Stokes, found in 3578 cases examined as to pulse-rate that an unusually slow pulse existed in six cases. In about 0·17 per cent of these cases, therefore, the condition was to be regarded as physiological. The pulse-rate in males is on the whole slower than in females, and all Grob's physiological cases occurred in men. Of persistent cases of bradycardia associated with signs and conditions which remove them from the category of the physiological, the majority also occur in men, although the condition is met with among women. In thirty-eight cases

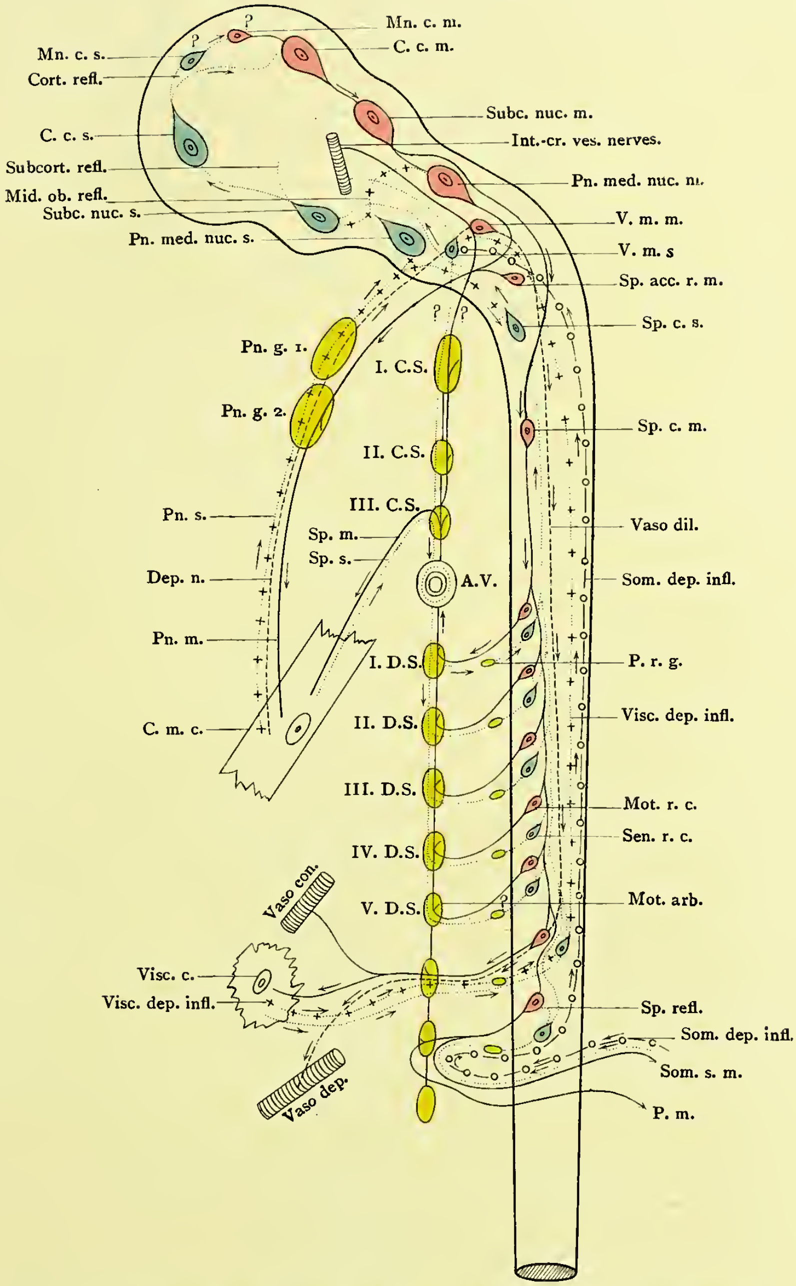


FIG. I.—DIAGRAMS OF VISCERO-NEURAL RELATIONS OF THE HEART.

Mark of interrogation attached to any letter indicates the uncertainty of the fact; pointed-out arrows indicate the direction of the travelling influence.

C. m. c. Cardiac Muscle Cell.
Pn. m. Motor portion of Pneumo-gastric Nerve.
Pn. s. Sensory portion of Pneumo-gastric Nerve.
Pn. g. 1. Root Ganglion of the Pneumo-gastric Nerve.
Pn. g. 2. Trunk Ganglion of the Pneumo-Gastric Nerve.
Dep. n. Depressor Nerve of the Heart.
Pn. med. nuc. s. Sensory Medullary Nucleus of the Pneumo-gastric.
Pn. med. nuc. m. Motor Medullary Nucleus of the Pneumo-gastric.
Mid. ob. refl. Reflex in middle Oblongata.
Subc. nuc. s. Subcortical Sensory Nucleus.
Subcort. refl. Subcortical reflex.
C. c. s. Sensory Cortical Cell.
Cort. refl. Cortical Reflex or Reaction.
Mn. c. s. Sensory Mnemonic Cell.
Mn. c. m. Motor Mnemonic Cell.
C. c. m. Motor Cortical Cell.
Subc. nuc. m. Motor Subcortical Nucleus.
Int.-cr. ves. nerves. Intra-cranial Vescular Nerves.
V. m. m. Motor Cell of Vaso-motor Centre.
V. m. s. Sensory Cell of Vaso-motor Centre.
Sp. acc. r. m. Motor Root of Spinal Accessory Nerve.
Sp. c. s. Sensory Cell in Spinal Cord.
Sp. c. m. Motor Cell in Spinal Cord.
Vaso-dil. Vaso-dilator Tract.
Som. dep. infl. Somatic Depressor Influence.
P. r. g. Posterior Root Ganglion.
Vis. dep. infl. Viscero Depressor Influence.
Mot. r. c. Motor Root Cell.
Sen. r. c. Sensory Root Cell.
Mot. arb. Motor Arborescence in Sympathetic Ganglion.
Sp. refl. Spinal reflex.
Som. s. m. Sensory-motor Somatic Nerves.
P. m. Pilo-motor Nerves.
Vaso dep. Vaso Depression.
Vaso con. Vaso Contraction.
Visc. c. Visceral Organic Cell.
Sp. m. Spinal Motor Nerve of Heart.
Sp. s. Spinal Sensory Nerve of Heart.
I. C.S. First Superior Cervical Ganglion.
II. C.S. Second Cervical Ganglion.
III. C.S. Third Cervical Ganglion.
A.V. Rings of Vicusseus.
I. D.S.–V. D.S. First to Fifth Dorsal Sympathetic Ganglion.

Cerebro-Spinal Sensory Tracts marked in dotted lines . . . . . + ···· + ···· + ···· +
Cerebro-Spinal Motor Tracts marked in uninterrupted lines . . . - - - - - - - - - - -
Other Tracts specially marked thus . . . . o—o—o—o

collected or observed by the writer, thirty-one occurred in males. The preponderance of this sign among men brings it statistically in line with the greater prevalence of angina pectoris in men as compared with women. As regards the influence of age, among the cases examined by the writer, one occurred in a boy 15 years of age, two between 20 and 30, two between 30 and 40, seven between 40 and 50, six between 50 and 60, eleven between 60 and 70, seven between 70 and 80, and one between 80 and 90. There was thus an increased frequency in the occurrence of the condition as age advanced, for the seventh decade may be regarded as the usual term of human life. Although the data upon which this conclusion is based are not numerous, it seems probable that bradycardia may be regarded as a "disease" of middle and later life and preferentially affecting the male. The alleged causes of bradycardia are cardiac overstrain from sudden or great physical exertion, alcoholism, syphilis, fibrosis of the heart, or fatty metamorphosis of the myocardium with or without associated arterio-sclerosis, and antecedent Graves' disease with tachycardia. Well-marked bradycardia of a less permanent kind has also been known to follow febrile movement and exhaustion, such as that which accompanies or is due to pneumonia and influenza. The changes which occur in the female organism after accouchement are also at times associated with slow pulse. A quasi-physiological form is known to occur after prolonged fasting or starvation, while reflex influences and some poisons, such as opium, and under certain conditions, digitalis, account for other cases. Finally, in a considerable number of reported instances, no cause could be assigned for even fatal cases of bradycardia, and the heart is stated, somewhat hastily, to have been perfectly healthy.

Symptomatology. — Like cardiac failure in general, persistent bradycardia may be of fairly long or of short duration; it may be an acute or chronic process. Persistent bradycardia of short duration is of necessity fatal, otherwise it would come into the category of the transient form which sooner or later gives place to a normal pulse, and is due to temporarily debilitating causes. The fatal form of persistent bradycardia of short duration is in reality a recurrent syncope, and is not very frequently fully observed. When it is, however, it presents a very striking clinical picture. "At short intervals there is a sudden cessation of all pulsation—a complete and prolonged intermission in cardiac action—followed at first by slowly recurring pulsations, 18, 20, 30, 40 times in the minute, again followed by another complete cessation of pulse. This syncopal bradycardia may be varied by short periods of more accelerated pulsation up to 60 or 70 in the minute, followed again by a complete pause. The patient during these pauses may not lose consciousness and may be quite aware of their advent, which he may signalise by an alarmed shouting and attempts at deep inspiration. Respiration is generally accelerated, the adjuvant mechanism of breathing coming to the assistance of the heart. If unconsciousness supervene, it may be associated with an epileptiform seizure. These grave symptoms may eventuate in recovery, but frequently the attack recurs and ultimately syncope becomes complete and prolonged, and death closes the scene" (Morison, *Cardiac Failure*, p. 56, London 1897). A well-marked, acute and transient bradycardia may, however, as has been remarked, be observed after exhausting febrile conditions, but soon gives place to a pulse of normal celerity, and of low tension, which in time improves in tone with the general recuperation of the patient (Figs. 1 and 2).

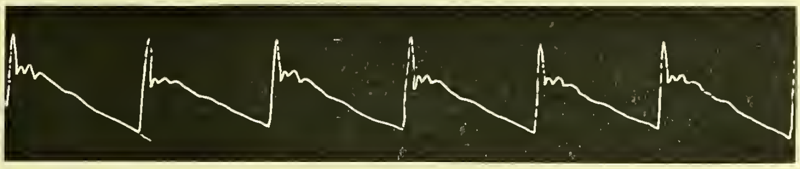


Fig. 1.—Bradycardia following pneumonia.

Fig. 2.—The same pulse soon after the bradycardia had disappeared.

Chronic persistent bradycardia may last for years, and is frequently associated with a systolic bruit at the aortic orifice due to atheromatous or sclerotic changes in that situation. The pulse may be slow and full, and in correspondence at wrist and heart (Fig. 3); or, a slow bigeminal pulse may have its dropped-beat phase so little marked as to be scarcely perceptible at heart or wrist, so as to convey the impression, without careful observation, that the pulsations

Fig. 3.—Chronic persistent bradycardia.

are half as numerous as they really are (Fig 4). True persistent bradycardia is inaccelerable by posture, exercise, or stimulation. The rate of respiration may be slightly diminished when bradycardia is equable and persistent, but is

Fig. 4.—Bradycardia with dropped beat.

usually accelerated more or less when the condition is due to cardiac failure of a most acute character.

Diagnosis.—Bradycardia being a mere symptom is not to be distinguished from any other condition. But two points have to be borne in mind in examining such cases, namely, (1) the possibility of cardiac action and pulse-rate manifesting the sesquipedalic or dropped-beat phenomenon, a point which must be determined by an examination both of the heart and the radial pulse; and (2) the fact that bradycardia may be due to causes either within or outside the heart. To determine the latter point Dehio has ingeniously suggested that atropine may be employed as a test. In intrinsic, or, as Dehio terms it, cardial bradycardia, atropine fails to accelerate the heart, while in extrinsic or extra-cardial bradycardia it succeeds in doing so.

Pathology.—The pathological conditions which have been found in association with bradycardia are proliferation of the interstitial tissue of the heart, fatty degeneration of its muscle cells, atheromatous changes at the aortic orifice with, in many cases, systolic bruit, and likewise, in many cases, in association with a general arterial thickening, usually due to senile change. The defective quality of blood so frequently found in connection with fatty degeneration of the heart and various cardiac neuroses may also be present when the neurosis takes the form of bradycardia. Visceral nerve changes, central and peripheral, have been little investigated. In Holberton's well-known case, the autopsy in which was made by Robert Liston, there was said to be some induration of the medulla oblongata, and enlargement of the pneumogastric nerves as well as of the middle cervical ganglion on the right side. In congested hypertrophic conditions of the heart Ott has found proliferative changes in the cardiac ganglia, and in dyscrasiæ due to anæmia and pyæmia, fatty change in the ganglion cells themselves, together with a like metamorphosis of the muscle cells. It is difficult, however, to attribute bradycardia to any one or to any combination of these changes, for some of them are even more frequently associated with tachycardial and arrhythmical conditions of cardiac action. Stokes of Dublin, who noted the frequent occurrence of bradycardia with obstructive atheromatous states at the aortic orifice, considered that the whole bodily conditions of which atheroma and arterial degeneration are the outcome, might be regarded as causal of slow pulsation. That the aortic lesion may have some influence in producing this state may, however, be argued from the comparative slowness and regularity of the heart's action in aortic as compared with mitral disease. Our ignorance of visceral neural pathology prevents the expression of any useful opinion as to the share taken in this matter by the nervous system, but there are circumstances which point to the abolition or depression of the accelerant action of the spinal cardiac nerves, however induced and associated with whatever anatomical changes, as the probable cause of retarded cardiac action.

Prognosis.—This in bradycardia due to fever, poisons, or sudden failure of the heart may, of course, not be gloomy. The effect passes off with the disappearance of the cause. But in persistent cardial bradycardia the prognosis is always grave. The usual termination is in death by syncope with or without associated epileptiform phenomena.

Treatment.—The treatment of bradycardia must be guided by the view taken of its most probable cause, the opinion formed as to its transient or persistent character, and the possibility of its being influenced by therapeutic agencies. Extra-cardial bradycardia must be combated by cardiac accelerants, of which belladonna, ether, and the nitrites are the chief. The bradycardia due to sudden dilatation or cardiac overstrain may be beneficially influenced by rest, the digitalis group of remedies, and in some cases by bleeding. Persistent bradycardia in elderly people, however, which, as a rule, is uninfluenced by posture or stimulants, calls for little treatment beyond that proper to senile arterio-sclerosis in general. Chief among these may be mentioned the employment of short courses of mercury, with or without the addition of digitalis or belladonna, and an occasional saline aperient. In many cases, however, a "masterly inactivity" is indicated. If the bradycardia be unassociated with subjective discomfort, it is wisest in many cases to avoid the use of specific cardiac agents, which may upset the balance, if they act at all, of what is in reality a form of established cardio-vascular compensation. The issue of such cases being as a rule syncope, emotional and physical stress, and the sudden increase of the vascular contents by the imbibition of large quantities of nutritive fluids, should be avoided.

Tachycardia.—The term tachycardia was first applied by Gerhardt to a markedly quickened action of the heart which had been previously described in this country. Accelerated cardiac action may be met with in various degrees and

continue for very varying periods of time. So-called "palpitation of the heart" is usually a transient acceleration of the heart's action, but being usually of short duration, and the acceleration in many cases not excessive, it has not been classed with tachycardia proper, of which at least three varieties exist. These are: (1) A considerable acceleration associated with certain nerve-lesions, to which reference will be made again; (2) A long-continued acceleration without such lesions; and (3) An excessive acceleration of comparatively short duration, arising suddenly and, as a rule, ceasing as suddenly, and usually associated with a detectable degree of cardiac dilatation, to which Bouveret in 1889 applied the term essential paroxysmal tachycardia.

The influence of sex is more evenly divided between the male and female in tachycardia than in bradycardia, and its commencement or occurrence in earlier life than the latter condition has also been remarked. The condition may, however, be met with in its most typical and paroxysmal form in comparatively advanced life, as in the fifth or sixth decade. These facts appear to argue its essentially neurotic character, as the nervous system of the child and of woman are more mobile than that of man as a rule, and the catabolic or quickened action of the heart is, on the whole, a more frequently-witnessed physiological phenomenon than anabolism or retardation. Neurasthenia, or that gradual and general loss of reserve force in the nervous system which is usually associated with exaggerated reflexes, mental and bodily, is particularly conducive to paroxysmal outbursts of accelerated cardiac action. Cardiac overstrain may, in this case, as in the case of other forms of disordered cardiac motion, be an important causal factor. External agencies, such as belladonna and tobacco, may likewise induce the condition. Tachycardia is indeed of the belladonna type of poisoning, as bradycardia is of the opium type. Certain cardiac valvular defects likewise conduce to the occurrence of tachycardia. It has been remarked that bradycardia may be associated with aortic lesions, and that aortic lesions are generally associated with less acceleration than mitral lesions. The mitral type of cardiac action is, on the other hand, tachycardial, and we shall learn that cardiac dilatation, with mitral insufficiency, is not infrequently observed in paroxysmal tachycardia. The fact that one of the functions of the pneumogastric nerve is cardiac inhibition, and the fact, likewise, that pressure upon these nerves by neoplasms or other growths is frequently associated with a measure of tachycardia, have led to the maintenance of the "vagus theory" of the condition, the action of that nerve being assumed to be more or less hampered, or in abeyance, under these circumstances. It must be remembered, however, that both physiologists and surgeons have shown that injury of one of these nerves has little permanent effect upon the heart's action, while injury of both is usually soon fatal. Herringham inclines to the opinion that not the nerve as a whole, but its peripheral endings, are at fault in tachycardia. Others have regarded it as an affection of the sympathetic nerves of the heart, and yet others as due to disease of the myocardium. Regarding the muscle cell as the ground common to the nerve-endings both of the vagus and sympathetic nerves, and as having a rhythmicality in a measure independent of either, it seems possible that there may be an emancipation of cellular rhythmicality from all nerve control in excessive paroxysmal cases; while in others, characterised by acceleration and augmentation, or by increased force with less rapidity, that the catabolic nerves may be active agents in producing the condition, the action of the retardant nerves being for the time in abeyance, but accumulating that energy which at last suddenly asserts itself in the restoration of slower action to the heart. We must be on our guard, however, not to throw the reins on the neck of imagination when we enter that area of neural exposition which has been termed the refuge of the destitute in search of theories.

The occurrence of such disorders in children whose organic cells are presumably normal, and the probable dependence of organic irregularities in some cases on reflex causes outside the organ most notably affected, seem to leave the initiative in such processes to the nervous system. Hence the justification for regarding these conditions as neuroses.

Symptomatology.—An accelerated palpitation of the heart with a sense of "fluttering" in the præcordial region, coming on without observable cause or after slight exertion, is a common phenomenon in anæmic persons, usually young, and as usually females. While this common condition is not that which has been raised to the dignity of a disease by the style and title of tachycardia, it is, nevertheless, a humble attempt in that direction. Could it succeed in persisting longer—for days, months, or it may be years, with an average pulse-rate of 120 or 130 in the minute, without other observable disorder, the condition would constitute persistent tachycardia. This state may for long be associated with little subjective sense of physical discomfort or general functional inactivity, and be indicated by no other fact than a persistent rapidity of pulse, perhaps accidentally detected. In course of time, however, even these cases tend to give out, and the tachycardiac patient becomes unfitted for the exertion hitherto undergone with apparent impunity. Functional disturbances of secretion and excretion manifest themselves, and sleep, the great recuperator of the nervous system, may become defective and unrefreshing.

If, as may happen, organic disease of the nervous system, peripheral or central, and due to intrinsic changes or external pressure, be associated with the tachycardia, the evidences of these may in time become more apparent. The third and highest degree of tachycardia, being associated, after much shorter duration, with considerable subjective discomfort, is that which Bouveret termed essential paroxysmal tachycardia. The pulse-rate is usually about 200 in the minute, and can only be counted by auscultation of the heart (Fig. 5). It may continue for periods varying from a few hours to several days, and the longer the duration of the attack, the greater the probability of the occurrence of retrograde phenomena, such as congestion of the lungs, enlargement of the liver, and albuminuria. It is indeed probable that attacks of tachycardia, unassociated with much or any subjective sense of palpitation, may cease and only leave the retrograde phenomena as evidence of their recent occurrence. It is in this way that the writer is inclined to explain the occurrence in a patient of his of consolidation of the base of the right lung, prune juice expectoration, and very slightly-increased respiratory rate, but without either pyrexia or acceleration of pulse. The patient, a lady about thirty years of age, informed him that she had felt faint and giddy the day previous to his detecting these signs, but had had no other symptoms pointing to her condition. She was of a neurotic constitution, and suffered much from urticaria.

Herringham observed in a girl of eleven years, the subject of an attack of essential paroxysmal tachycardia, that the apex-beat was more visible than usual, in addition to the rate of pulsation being greatly increased.

Percussion usually shows the cardiac area in these cases to be increased in extent, and the sphygmograph declares the small and quick

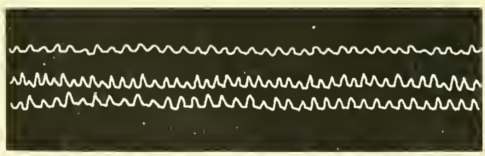


Fig. 5.—Radial pulse of essential paroxysmal tachycardia.

pulse to be one of low tension. Prior to the occurring of secondary stasis, there may be no subjective distress beyond a general sense of weakness, and the attack may subside as suddenly as it originates, leaving a pulse of normal tension and rapidity (Fig. 6). Herringham remarks that the attack often terminates during sleep, but that it has been determined to continue unabated during sleep in persistent cases.

Diagnosis.—The transient tachycardia so frequently met with among anæmic patients is easily distinguished by the presence of other signs of anæmia, and by the removal under treatment of the symptom with the improved condition of the blood. A persistent tachycardia of moderate rapidity may be due to commencing Graves' disease, in which the classical signs of

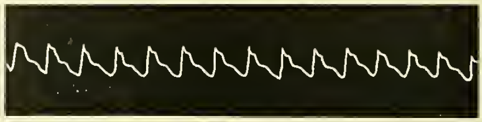


Fig. 6.—The same pulse as Fig. 5, after the attack had subsided.

that condition have not as yet developed, or in which one or other of the striking phenomena in eye, or thyroid, or muscular tremor, are long delayed or altogether absent. Rapid arrhythmical pulsation of the heart may be distinguished from tachycardia proper by its very arrhythmicality, and the usually associated signs of arterio-sclerosis, which are frequently altogether absent in tachycardia, and also by the amenability of rapid arrhythmiæ to treatment. With essential paroxysmal tachycardia no other condition can be confused, nor is the degree of sudden cardiac dilatation associated with that state encountered in any other condition, in which the heart muscle gives evidence of general soundness.

Pathology.—The transient tachycardia of anæmia has no recognisable physical basis beyond the characteristic alterations in the blood met with in that state, any more than has the neuralgia associated with the same condition. The hypersensibility conferred upon the sensory portion of the nervous system by anæmia, and manifested in some cases by neuralgia, is shown in yet others by disordered motor innervation, and by exalted irritability of rhythmical cells. In sustained tachycardia of moderate rapidity (100 to 150 beats in the minute) tumours in or pressing upon the pneumogastric have been met with in some cases, and in two mentioned by Martius both pneumogastrics were involved. As, however, in the latter cases the patients lived for some time in this condition, it is not probable that the conducting power of the nerves was altogether abolished. In bulbar paralysis, also associated with tachycardia, the pneumogastric nucleus has been known to be invaded by disease. Besides these changes, in five cases quoted by Herringham, there was fibrosis of the left ventricle in two, fatty degeneration of the myocardium in one, while in the remaining three dilatation was the only abnormality discovered.

Prognosis.—The prognosis in tachycardia, as in bradycardia, depends upon the nature of the cause. If this be removable the effects may also be expected to disappear. The cure of

anæmia will also cure such tachycardia as is dependent upon it, the removal of sources of reflex tachycardia will likewise benefit the latter, but in a large proportion of the persistent cases the prognosis is not favourable, although the fatal issue may be delayed for a considerably longer period than is usually the case in "cardial bradycardia." The association of tachycardia due to any cause with organic valvular disease of the heart renders the prognosis both of the neurosis and the mechanical lesion doubly unfavourable, and the end may be sudden and syncopal, or more gradual and associated with all the retrograde venous phenomena of progressive cardiac failure.

Treatment.—In the treatment of the tachycardia symptomatic of anæmia, the use of arsenic is advisable, in addition to any other hæmatinic, such as iron, because there is reason in the belief that it has a tonic influence upon the nervous system. The peripheral palsies, induced by its over-use, argue a certain stimulating influence on peripheral visceral nerves when it is carefully employed. The drug treatment of persistent or paroxysmal tachycardia has, however, not been found to be effectual. Nevertheless, pharmaceutical agents calculated to support the three factors in organic action—the blood, the nerves, and the muscle cell—should be carefully and persistently employed, in conjunction with such hygienic measures as it is within the power of the patient to avail himself of. The representatives of this line of treatment may be said to be arsenic, iron, the digitalis group of remedies, the bromides, and, in the instance of cases of specific origin, the iodides and mercury.

In paroxysmal cases associated with valvular lesions, and which are not, of course, as a rule, cases of "essential tachycardia," much may be done, as we all know, by the recognised treatment of the lesion, for particulars of which the reader is referred to p. 136 *et seq.* The effect of opium in some of these cases, the physiological influence of which is bradycardial, is very striking.

Among external agencies a word may be said about the employment of cold. The careful employment of cold, in the form of ice, to the head or præcordia, for such periods as the patient can tolerate it, is frequently of service. A course of Nauheim baths is at times said to be of service, but the addition to this of gymnastic exercises, whether administered manually or by mechanism, is not to be undertaken without the closest supervision by the physician himself. Certain positions of the body, accidentally or instinctively assumed by the patient, appear at times to bring about improvement. Thus, compression of the thorax and abdomen and flexion of the legs on the trunk, have appeared to be of service, and probably act by raising the peripheral blood-pressure. Herringham recommends that pressure on the vagus should be always tried, "although it seldom succeeds." It is difficult, indeed, to see why it should succeed unless both vagi be compressed. We know that intermission of the heart's action may at times, and usually under special circumstances, be induced by compression of the vagus, but a vigorous and bilateral employment of this method does not seem to be free from the possibility of injurious interruption of the cardiac cycle. The treatment of known sources of reflex stimulation of the heart's action, such as local inflammatory disease of any kind, and displacement of organs, such as floating kidney, is indicated. The removal of adenoids is reported to have been curative in one case (Spencer Watson).

Having said so much, however, it ought to be added that it seems as impossible to retard the action of the heart in many cases of tachycardia, persistent and paroxysmal, as it is to accelerate it when bradycardia is of long duration. They appear to be what Dehio would call cardial, not extra-cardial, in origin, and, as such, unaffected by the agencies which, under other circumstances, influence the action of the heart.

ARRHYTHMIA.—Under this term are included all abnormalities of cardiac action not solely characterised by diminution or increase of the rate of pulsation. In some respects the term is misleading, for abnormalities in pulsation are frequently observed which, while they differ from the isochronism of normal action, are, nevertheless, rhythmical in their arrhythmia. To rectify this contradiction, however, would lead to a multiplication of terms, which it is advisable, so far as is possible, to avoid. An ideal nomenclature no doubt would convey not merely the recognition of the fact of a particular kind of abnormality, but likewise the physiological nature of the action in question, and this the sphygmograph enables us in some measure to do in these cases.

Using arrhythmia in its widest sense, the condition is met with at all ages from childhood to old age and in both sexes. Its persistence, however, for a sufficient length of time to entitle it to be regarded as a definite morbid condition, is most frequently met with in adult life, and especially after the meridian of life is past. That is, when the wear and tear of life leaves its traces in texture. The condition is more common than either persistent bradycardia or persistent tachycardia, and perhaps owing to this very fact a statistical estimate of its incidence at certain ages and in both sexes is difficult to obtain. The writer believes, however, that it may be asserted that persistent arrhythmia, unassociated with organic valvular disease of rheumatic origin, is more common in men than in women, and most common among those in the arterio-sclerotic period during and after the fifth decade. The arrhythmia associ-

ated with rheumatic valvular disease may of course be met with much earlier and at any age, although, inasmuch as the same forms of arrhythmia may be met with both in the presence and in the absence of valvular lesions, the latter cannot be regarded as essential factors in their production.

The nature of the lesion appears, however, to have a certain influence upon the character of the heart's action in its endeavour to cope with the mechanical difficulty produced by it. Thus a certain type of arrhythmia may be more common in one form of valvular lesion than in another. Just as the heart's action is usually more regular when compensation is lost in aortic than in mitral valvular disease, so the arrhythmia associated with aortic disease tends to be more rhythmical and of a slower rate than the delirious hyposystolic pulsation of a tachycardial type so often met with in mitral disease (Figs. 7 and 8). What applies to the aortic and mitral valves applies also to the pulmonary and tricuspid, although the opportunity of observing the latter is rarely afforded. There may therefore be said to be an aortic and mitral type of arrhythmia, the former having a greater leaning towards bradycardia and the latter towards tachycardia, with the result that the arrhythmia of necessity partakes of the nature of both, periods of retarded pulsation occurring irregularly in the midst of an unequal tachycardia. Outside mechanical causes, the same influences which provoke bradycardia and tachycardia have an influence in the production of arrhythmia. Gout in its protean forms—rheumatism, influenza, alcoholism, syphilis, venery, tobacco, near or distant local causes of reflex action, and interstitial sclerotic and myocardial changes, may be associated with this as with other forms of disordered cardiac action. The discrimination of these is, however, of practical importance, as will be insisted upon in its proper place. Finally, when predisposing causes are present, "shock" may originate a persistent arrhythmia. Nor need this surprise us when we reflect that both physical and mental shocks may induce a fatal syncope and indeed persistent disorder in organs other than the heart. Thus a neurotic patient of the writer's, when towards the end of his fifth decade and in good health, developed glycosuria as the result of the suicide of an intimate friend whose cremation he had to superintend in accordance with the wishes of the deceased, and about five years later, having the misfortune to be knocked down in the street by a bicycle going at good speed, the same subject developed a persistent and ultimately well-marked arrhythmia, which dated from that accident. In notes made prior to that event, his pulse was only on one occasion observed to be slightly intermittent (Figs. 9 and 10).

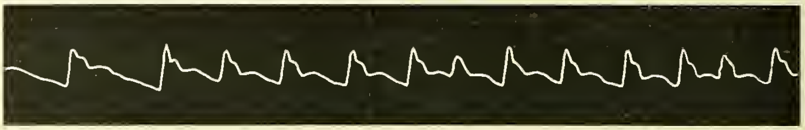


Fig. 9.—Incipient arrhythmia after shock.

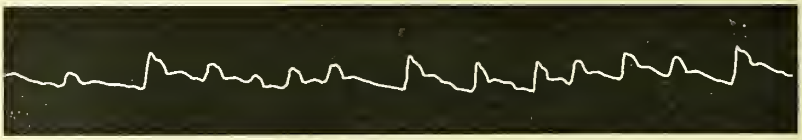


Fig. 10.—The same pulse four years later.

Fig. 7.—Rhythmical arrhythmia in aortic valvular disease with trigeminal pulse.

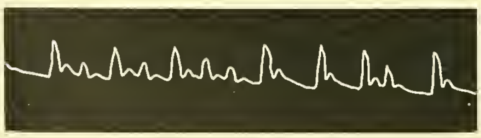


Fig. 8.—The arrhythmia of mitral disease.

Symptomatology.—The inequality in *time* in the pulse of arrhythmia is usually associated with a variety in the *character* of the separate cycles in cardiac action and their corresponding pulse waves. In the simplest form of arrhythmia, indeed, the variation in time is more marked than in character (Fig. 9), but sooner or later alterations occur in this respect also. The pulse wave may then be characterised by a partial systolic rise which corresponds to a hyposystole at the heart, by a bold intercurrent systolic rise which denotes a hypersystole at the heart, or by a suppression of the systolic rise or intermission of the pulse, which denotes the condition of asystole at the heart (Fig. 10). All these may, moreover, be met with in the same pulse, and occur without determinable periodicity. The arrhythmia is then complete. Systole in varying force may, however, be associated, as has been stated, with hyposystole

of regular recurrence, and give rise to the cardiac and radial pulse evidences of coupled and tripled beats (Figs. 4 and 7). In some cases the patient, when recumbent, evinces the coupled beat at the wrist and a quadruplication of sounds at the heart, which may be rendered phonetically as lūp—dŭp—lūp—dūp, and on assuming the erect position, the heart's action being quickened, the pulse becomes regular at the wrist and the cardiac sounds may be rendered as lūp—lūp—dūp. The acceleration of the heart in this case obliterates the first diastolic pause in the coupled beat, and the change is marked by an anachrotism of the pulse not previously present (Figs. 11, 12, and 13).

The objective signs of arrhythmia may be associated with no subjective discomfort on the part of the patient. On the other hand, a forcible irregularity of the heart may give the sense of palpitation, and well-marked intermissions are also frequently felt by the patient at the moment of their occurrence. The heart is felt to "stop," as patients at times remark. It is unnecessary to state that these objective and subjective signs may also be associated with other evidences of cardiac failure, local and general, such as valvular bruits, and various somatic and visceral forms of venous stasis. They may also manifest themselves without any of the notable signs of cardiac debility.

Diagnosis.—Arrhythmia, as its name denotes, is essentially *irregular pulsation.* It may be too quick a pulsation of the heart, with intervals of slower pulsation, and this *irregularity* is one of the means of distinguishing it from tachycardia. It is never likely to be confused with bradycardia, although in some cases, as has been stated, it may give place to a persistent domination of the latter sign. The diagnosis of tachycardial arrhythmia from persistent tachycardia may also be made by observing the effects of treatment by posture and drugs. The heart-rate of the former is more certainly reduced than that of the latter. The discrimination of tachycardial arrhythmia with functional bruit, usually soft and mitral systolic, from the same condition in association with organic valvular disease due to rheumatic endocarditis, is also important. As Huchard has pointed out in the former case, the bruit is a consequence of the disorder of cardiac motion and late in appearing, while in the latter the bruit is of early occurrence, and usually noted before the arrhythmical phenomena manifest themselves. In many cases, moreover, the functional bruit is due to mitral insufficiency from cardiac dilatation of a temporary kind, while the organic naturally persists, however much the heart may receive muscular compensation. Indeed, the more compensated the heart becomes, the more audible, in many organic cases, is the bruit.

Pathology.—The ultimate disappearance of arrhythmia in many cases, and at various periods of life, leads to the conclusion that it may sometimes depend upon transient conditions affecting one or more of the three factors in organic action, which do not leave permanent and recognisable traces in the heart. As, however, a large proportion of persistent cases occur within the arterio-sclerotic period of life after the fifth decade, it is not surprising to find that

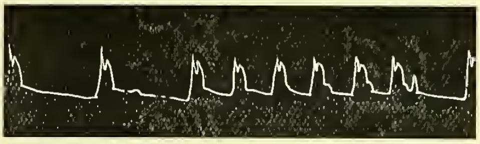


FIG. 11.—Arrhythmia with asystole.

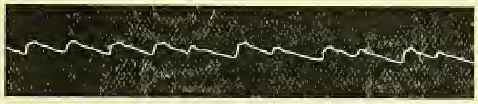


FIG. 12.—Arrhythmia with quadruplication of heart sounds when recumbent.

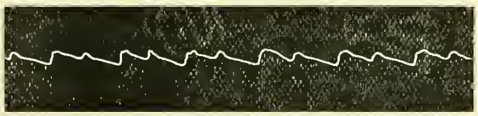


FIG. 13.—Another of the same.

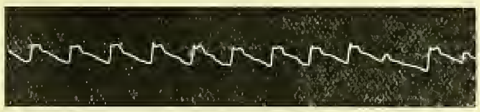


FIG. 14.—Triplication of heart sounds in the same patient when erect.

interstitial and myocardial changes, fibrosis, pigmentation, fatty degeneration of the heart, together with more general arterio-sclerosis, are associated with many cases of persistent arrhythmia. Inasmuch, however, as all these states may occur in an aggravated degree, and with every phase of cardiac failure, without the patient manifesting arrhythmical action of the heart, it follows that causes still largely unexplained lie at the bottom of the disordered action of the heart.

Of the intimate pathological changes in cardiac innervation, and in the innervation of other viscera, little more is at present known than has been already mentioned, but the more systematic examination of this point after death, and the more regular examination of the blood in such cases during life, are calculated to afford useful and much-needed information. There can be little doubt that the toxins generated by the germs of such infectious diseases as influenza may exact a morbific influence upon the visceral

innervation, although of a less easily detectable character than the gross neural changes met with after diphtheria.

Prognosis.—Reflex and toxic arrhythmia may disappear at any age with the disappearance or removal of the cause, as likewise may the arrhythmia of cardiac overstrain and general exhaustion when these states arise in comparatively young people with a fair reserve of recuperative energy. The arrhythmia of the arterio-sclerotic age is, on the other hand, usually persistent and progressive, inducing cardiac dilatation and other evidences of cardiac failure, and tending to shorten the life of the patient. Under appropriate treatment, however, much may be done to prolong life in these cases, even when the underlying pathological condition is in an advanced stage, and the signs of cardiac failure well marked.

Treatment.—In this as in other forms of disordered cardiac action the removal or restraint of sources of reflex irritation is indicated. In the case of the young these will usually be found to lie in the gastro-intestinal tract, and will be amenable to the usual dietetic and medicinal agents effectual in securing due digestion in and evacuation of the canal. In other cases, the treatment of a general condition of which the arrhythmia is but a symptom, is indicated. Thus, in the writer's experience, a case yielded to the use of thyroid extract in the case of a lady, the subject of indistinctly-marked myxœdema. The gout, glycosuria, or rheumatic heart affection associated with arrhythmia calls for treatment on recognised lines, for particulars of which the reader is referred to other portions of this work.

If arrhythmia be associated with obesity or neurasthenic conditions suggestive of impaired muscular power, a judicious alternation of rest and exercise, with or without salt baths, on the Nauheim principle, may be of much service. It is, however, in cases of arrhythmia associated with arterio-sclerosis that careful management may effect much. In these the Nauheim system, although often employed, is of little service, and not devoid of danger. These cases, in a large proportion of mankind, need cardio-vascular rest, not exercise, but rest need not be synonymous with absolute quiescence, unless well-marked evidence of cardiac failure be present. A regulation of the blood weight by prescribing small and easily digestible meals, and the avoidance of more than a minimum of alcohol and tobacco, together with gentle exercise, may do much among the better classes to promote efficient cardiac action. The question as to the advisability of absolutely prohibiting the use of tobacco frequently arises. In the case of those who have undeniably over-smoked themselves, it may be as necessary to do so as it may be to absolutely prohibit the use of alcohol to the drunkard. But the rule of thumb and oracular prohibition of these articles may, in the case of those accustomed to their moderate use, do more harm than good. Although the will cannot influence the heart, the emotions can, and a large number of people cannot reconcile themselves to the abandonment of these articles without an amount of subjective worry, which it is desirable to avoid when the controlling influence of mental calm is very desirable. The poor overworked arterio-sclerotic patient, with arrhythmia, on the other hand, frequently does well while in hospital on ordinary diet, rest in bed, and two to three ounces of brandy daily. He needs feeding, rest, and warmth to strengthen his heart and to overcome his peripheral vascular resistance. Among drugs in such cases, if one be more valuable than another, it is mercury, given alone, or, if there be evidences of cardiac dilatation and failure, in combination with digitalis. Old masters, such as Stokes of Dublin, knew well the value of this combination, and that value will be found not to have been exaggerated by those who will follow their directions in the present day.

The proper use of mercury in arterio-sclerosis seems, indeed, for a time to have been largely a lost art. With the concurrent use of bland unirritating form of food, and the avoidance of acid condiments such as vinegar, however, mercurials may be given for a considerable period with benefit. In arterio-sclerotic cases with arrhythmia and cardiac failure, the well-known combination of blue pill, digitalis, squill, and hyoscyamus, often known as the "Guy's Pill" (but which is really attributable to that worthy physician, Dr. Matthew Baillie, who had no connection with Guy's Hospital), acts in many instances with the efficacy of a specific. The combination, indeed, often succeeds when the ingredients given separately fail, as Dr. Baillie maintained, for, while the mercury relieves the peripheral resistance to the circulation, the digitalis urges the weakened heart to more efficient action.

Heart, Surgery of.—As surgeons have advanced from the surgery of the pleura to the surgery of the lung, so the advance has now been made from puncture or incision of the pericardium to suture of a wound of the heart, and even to ligature of a bleeding coronary artery. The necessity for such an operation will very seldom come, yet a surgeon ought to have in his mind how he would meet it.

To clear the ground, those cases may be set aside where a needle or a knitting-needle has been driven by accident, or in an attempt at suicide, into the heart. In these cases the rule is clear and unmistakable, that the *foreign body* should be at once removed. One end of it may be felt under the skin; or its exact position may be defined by the X-rays. Even though it be gone altogether inside the pericardium, yet

it should not be left there, unless it be so small that it might not be found after incision of the pericardium. In most of the published cases, it has not been necessary to do more than incise the superficial structures; nor has any serious bleeding followed the withdrawal of the foreign body.

Again, cases of *gunshot wound* need not here be considered; for though a case of surgical interference has been published by Riedel, and another by Podrez, yet such an operation must ever be one of the rarest possibilities of surgery; nor did their methods of procedure differ in any material way from those that have been used in cases of punctured or incised wounds made with a knife.

It is these *stabs* over the heart that have offered most opportunity for treatment by operation—the cases of Farina, Cappelen, Tassi, Rehn, Parrozani, and others. And, before operating, the surgeon has to face the fact that some undoubted cases of wound of the heart have recovered without operation. The older methods of treatment, absolute rest, without moving or speaking, real "immobilization," ice applied over the heart, low diet, and copious venesection—these methods did sometimes succeed. Nothing but hourly watching, and seeing evidence of steadily increasing pressure on the heart, and blood accumulating in the pleura, and the patient going in spite of treatment from bad to worse, justifies operation.

In a case of stab-wound of the heart, it is not likely that the surgeon need be afraid of wounding the pleura. Practically, he may be certain that he will find it already opened, and with blood poured into it. The internal mammary artery he may find divided, or may divide it, or may not come across it. The incision through the skin and the muscles has been made different ways, according to the position of the wound; either a long curved incision, or a flap. The cartilages resected have been, in most cases, either the fifth, or the fourth and fifth together. And, by whatever method, the surgeon must work freely, through a space large enough to let him see what needs to be done.

It has been recommended that the wound in the pleura should be at once closed, and the pleura pushed outward, out of the way. But, in practice, the opposite has been done; the wound in the pleura has been freely enlarged, the blood and clots have been washed out so thoroughly as the case permitted, and the pericardium has been dealt with through the pleura. The surgeon has found the wound of the pericardium, with blood running out of it, has enlarged it, and secured its edges with catch forceps.

There are, of course, cases where the pericardium has been opened, and no wound found on the heart (Tassi, Williams, Dalton). In Mr. Mansell Moullin's case (*Lancet*, 1897, i. 314) the patient had been kicked over the heart; the pericardium was opened, found full of blood, and drained, and he made an excellent recovery. In a few cases a wound of the heart has been seen, but so small as not to need suture.

In other cases the wound has been sutured with fine silk and a fine curved needle. From one to four sutures have been placed; and the ends of the first suture have been lightly held, to make it easier to pass the next. Cappelen passed, and tied, his sutures during the systole of the heart; Rehn, during its diastole.

In some cases the pericardium has been drained; in some, it has been closed at once. The hæmothorax may give rise, many days afterward, to the need of an operation to drain the pleura.

Heat. *See* Balneology (*Effects of Heat*); Disinfection (*Heat as a Disinfectant*); Fœtus and Ovum, Development of (*Temperature of Fœtus*); Invalid Feeding; Medicine, Forensic (*Burns, Heat-Stiffening*); Physiology, Food and Digestion (*Production and Elimination of Heat*); Sunstroke; Tropics, Unclassed Fevers of (*Heat Apoplexy, Insolation*); Ventilation and Warming.

Heat Fever. *See* Sunstroke.

Heating. *See* Ventilation and Warming.

Heat Stroke. *See* Sunstroke.

Heberden's Nodes.—Tubercles or small hard knobs which develop on the phalanges of the fingers in arthritis deformans, etc. *See* Gout (*Chronic Deforming Gout, Nodi Digitorum*); Joints, Diseases of (*Arthritis Deformans sicca*); Rheumatism, Rheumatoid Arthritis (*Monoarticular, Morbid Anatomy*).

Hebetude.—Dulness of intellect or mental obtuseness; *Hebetudoanimi* is imbecility. *See* Mental Deficiency.

Hebotomy.—Lateral section (by Gigli's wire-saw) of the pubes for the purpose of temporarily enlarging the pelvic brim (in cases of contracted pelvis) to allow of the passage of the fœtal head and trunk; the term is derived from the Greek Ἥβη, Hebe, the pubes being the bone of Hebe; the operation was revived by Gigli of Florence in 1902; pubiotomy or pubotomy. *See* Labour, Operations, Symphysiotomy.

Hectic Fever.—A condition in which the temperature rises daily (in the afternoon and evening) to 104° or 105° F., and is subnormal in the morning; it is associated with suppuration, tuberculosis, etc. *See* Suppuration (*Acute Circumscribed Abscess, Clinical Features, Constitutional, Pyrexia, Hectic*).

Hedonal.—A derivative of urethane, methyl-propyl-carbinol-urethan; used in doses of 15 to 45 grains as a hypnotic and analgesic; it was discovered by Dreser in 1889; and it has been employed in insomnia, in chorea, and before the inhalation of chloroform (in order to lessen the amount of chloroform necessary to produce anæsthesia).

Hedonism.—"Mental eccentricity characterised by the following of some special pursuit in an unreasoning manner" (*Hack Tuke*).

Heel-Jerk.—The Achilles-tendon-phenomenon, elicited by tapping the tightly-stretched tendo Achillis.

Hegar's Dilators.—A series of graduated rubber (or metal) rods, used for dilating the cervix uteri. *See* Curettage; Gynæcology, Diagnosis in (*Cervical Dilators*).

Hegar's Sign.—This sign of early pregnancy is elicited by bimanual examination of the uterus, two fingers of one hand being in the vagina and the other hand over the abdomen, or one finger being in the rectum and the thumb in the vagina; the marked softening of the lower uterine segment gives to the examining fingers the sensation as if the cervix and the body of the uterus were detached from each other. *See* Pregnancy, Diagnosis (*Softening of the Lower Uterine Segment*).

Hehner's Formula.—A means of calculating the amount of fat in milk from the specific gravity and total solids. The formula is as follows: $F = \cdot 86\ TS - \cdot 22\ G$ (F = fat, TS = total solids and G = specific gravity − 1000).

Height and Weight. *See* Children, Development of (*Weight and Length*); Fœtus and Ovum, Development of (*Length and Weight*); Life Insurance (*Height and Weight in Adult*).

Helalin.—Helalin is stated to be the active principle of *Collinsonia canadensis*; it possesses a sedative effect on mucous membrane, especially those of the urinary and gastro-intestinal tract (*e.g.* in cases of gonorrhœa, cystitis, renal and biliary colic); the dose is 1 drachm of the concentrated solutions prepared by Messrs. Oppenheimer.

Helco-.—In compound words *helco-* (Gr. ἕλκος, an ulcer) means relating to an ulcer; thus *helcology* means the science of the pathology of ulcers, *helcosis* ulceration, and *helcostaphyloma* ulcerative staphyloma.

Helcosoma Tropicum.—A protozoon organism found in the Delhi sore by J. H. Wright of Boston (1904).

Helenin.—A volatile oil (C_6H_8O), obtained by distillation from Elecampane root (*Inula Helenium*), and possessing antiseptic properties.

Helensburgh. *See* Therapeutics, Health Resorts (*Scottish*).

Heliotherapy.—The use of the sun's rays in the treatment of disease (*e.g.* in laryngeal tuberculosis).

Helix.—The outer border of the external ear (Gr. ἕλιξ, a spiral) or any spiral structure; *helicoid* means of a spiral form.

Hellebore.—The Black Hellebore (*Helleborus niger*) or Christmas Rose and the Green Hellebore (*Helleborus viridis*) both contain crystalline glucosides, named Helleborin ($C_{36}H_{42}O_6$) and Helleborein ($C_{26}H_{44}O_{15}$); the poisonous alkaloid *veratrine* ($C_{37}H_{53}NO_{11}$) occurs in White Hellebore (*Veratrum album*). *See* Toxicology (*Alkaloids, Hellebore*).

Heller's Tests. *See* Urine, Pathological Changes in (*Albuminuria*); Urine, Pathological Changes in (*Hæmoglobinuria*).

Hellopia. *See* Balneology (*Greece, Sulphurous Thermal Springs*).

Hellyer's Trap.—A ventilating and intercepting trap used in drainage; there is also a grease-intercepting *tank* called "Hellyer's."

Helmholtz Theory. *See* Accommodation (*Theories, Helmholtz*); Iris and Ciliary Bodies (*Physiology, Mechanism for Accommodation*).

Helminth.—An intestinal worm, *helminthiasis* being the condition of being invested by such worms. *See* Parasites (*Helminths*); Leucocytosis (*Eosinophile*).

Helmitol.—The anhydro-methylene citrate of hexa-methylene-tetramine; it has been used as a urinary disinfectant and antiseptic in cases of gonorrhœa, cystitis, etc., in doses of 15 grains thrice daily; it has also been recommended in scarlet fever as a prophylactic to prevent renal complications; it contains urotropin.

Helouan-les-Bains. *See* Balneology (*Africa, Egypt*); Mineral Waters (*Sulphur Waters*).

Helvella Esculenta. *See* Toxicology (*Food-Stuffs, Fungi*).

Hemaboloids.—A proprietary preparation, described as containing a synthetically-prepared true organic iron compound, nutrient albuminoids, the hematinic principle of bone marrow, and nuclein; it is recommended (in doses of half an ounce after food) for the purpose of increasing the number of red cells in the blood and the percentage of hæmoglobin.

Hemeralopia.—Night-blindness, or inability to see in insufficient light (twilight) or at night. This is the ordinary non-etymological meaning of the word (regarding it as synonymous with nyctalopia), but the correct signification on etymological grounds is day-blindness, or inability to see objects in daylight or in strong light, with ability to see them in twilight. The latter is the sense understood in the *Nomenclature of Diseases* (London 1906), and in Murray's *New English Dictionary*. *See* Choroid, Diseases of (*Congenital Affections, Albinism*); Iris and Ciliary Bodies (*Congenital Abnormalities of Iris*); Skin, Pigmentary Affections of (*Disappearance of Melanin, Albinism*).

Hemeraphonia.—Loss of voice during the daytime; usually hysterical.

Hemiacephaly. *See* Hemicephaly.

Hemiachromatopsia. *See* Vision, Field of (*Hemianopia*).

Hemialbumin. — A constituent part of albumin thought to be changed into *hemialbumose* during digestion; it may be found in the urine. *See* Digestion and Metabolism (*Metabolism of Proteids*).

Hemialgia.—Pain situated on one side only; unilateral pain.

Hemianæsthesia. — Unilateral insensibility or anæsthesia, met with in cerebral hæmorrhage (on the opposite side from the lesion), in unilateral cord lesions, and in hysteria. *See* Brain, Affections of Blood-Vessels (*Hæmorrhage, Localising Phenomena*); Brain, Tumours of (*Localising Phenomena, Sensory*); Chorea (*Symptoms, Sensory*); Hysteria (*Sensory Disorders, Anæsthesia*).

Hemianopia. *See* Amblyopia (*Varieties, Due to Changes in the Optic Nerve*); Brain, Physiology of (*Sensory Centres, Sight, Homonymous Hemianopia*); Brain, Affections of Blood-Vessels (*Hæmorrhage, Symptoms*); Retina and Optic Nerve (*Optic Nerve, Anatomy and Physiology*); Vision, Field of (*Hemianopia*).

Hemianopsia. *See* Auditory Nerve and Labyrinth (*Localisation of Lesion producing Nerve Deafness*); Brain, Affections of Blood-Vessels (*Paralysis with Sensory Disturbances*); Brain, Tumours of (*Localising Symptoms, Sight*); Hemiplegia. *See also* Hemianopia and its Cross-References.

Hemianosmia. — Unilateral loss of smell. *See* Nose, Nasal Neuroses (*Anosmia*).

Hemiathetosis. — Athetosis affecting one-half of the body. *See* Athetosis.

Hemiatrophy. — Unilateral atrophy. *See* Facial Hemiatrophy; Cheek, Fissure of (*Associated Malformations*).

Hemicephaly.—Anencephalus (*q.v.*).

Hemichorea. — Chorea affecting only one side of the body. *See* Chorea (*Symptoms*); Brain, Inflammations (*Encephalitis, Sequelæ*).

Hemicrania.—Unilateral headache or migraine. *See* Indigestion (*Special Forms, Hemicrania*).

Hemicraniectomy. — The exposure of one-half of the brain by dividing the vault of the skull near the middle line and pressing the side outwards (Doyen).

Hemidesmi Radix. — The root of *Hemidesmus indicus* or Indian Sarsaparilla; it is used for the same purposes as sarsaparilla; it contains tannin and hemidesmic acid (the supposed active principle); its preparation (*Syrupus Hemidesmi*) is given in doses of $\frac{1}{2}$ to 1 fl. dr.; but it is not generally regarded as having any marked pharmacological action.

Hemidiaphoresis. — Unilateral perspiration.

Hemignathus.—Congenital absence of one-half of the lower jaw.

Hemihypertrophy.—Unilateral overgrowth, or hypertrophy limited to one side of the body. *See* Facial Hemihypertrophy; Head (*Symmetry, Hemihypertrophy*); Hypertrophy.

Hemimelus.—The teratological type in which one or more of the four extremities ends in a more or less tapering point or stump resembling that left after an amputation. *See* Teratology.

Hemimyoclonus.—A unilateral clonic convulsion of the body.

Hemiopia.—Loss of vision over one-half of the retina; hemianopsia. *See* Pupil (*Nervous Mechanism, Hemiopic Pupil of Wernicke*).

Hemipagus. — Twins united by the heads and thoraces, having two faces, more or less incomplete, which are united laterally, and are turned more or less from the abdominal aspect; diprosopus monopedius. (*See* Ballantyne's *Antenatal Pathology*, ii. 632).

Hemiplegia.

See also Aneurysm (*Neck, Common Carotid, Pressure Symptoms*); Aphasia; Athetosis (*Etiology*); Brain, Affections of Blood-Vessels

(*Cerebral Embolism*, *Thrombosis*, *Hemiplegia*, *Alternate Hemiplegia*); Brain, Tumours of (*Diagnosis of position of Tumour*); Brain, Cerebellum, Affections of (*Hæmorrhage*); Diphtheria (*Complications*, *Paralysis*); Eclampsia (*Diagnosis*, *Complications*); Heart, Myocardium and Endocardium, Affections of (*Symptomatology*, *Cerebral Symptoms*); Joints, Diseases of (*Associated with Lesions of the Nervous System*); Malingering (*Nervous Diseases*, *Hemiplegia*); Measles (*Complications*, *Hemiplegia*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms*, *Nervous*, *Paralysis*); Neurasthenia (*Symptomatology*); Ocular Muscles, Affections of (*Paralysis*); Osteo-Arthropathies (*Mode of Production*;) Paralysis (*Infantile Hemiplegia*); Paralysis (*Pseudo-Bulbar Paralysis*, *Double Hemiplegia*); Typhoid Fever (*Complications and Sequelæ*, *Nervous System*).

Whatever objection may be raised to the name hemiplegia it is so widely used, and so easily understood, that no substitute, even were such forthcoming, is likely to displace it. By the term is understood a paralytic affection of the whole of one side of the body, with or without involvement of sensation, and clinical varieties of the ordinary condition depend upon the relative degree of the paralysis in the different parts of the affected side. Thus there is a condition in which the arm on the paralysed side is more affected than the leg; another in which the leg is more affected than the arm; another in which aphasia is superadded to the paralysis; another in which one half of each retina is also affected, giving rise to a condition of hemianopsia, and another in which the cranial nerves may be involved. These varieties depend upon the position in which the lesion causing the paralysis is situated.

Other varieties of hemiplegia are denoted by a descriptive title prefixed to them. Thus *crossed* hemiplegia (also called *alternate* hemiplegia) is a variety in which one side of the face (and perhaps also the external rectus muscle and also the sensation of one side of the face) is affected while the limbs on the *opposite* side are paralysed (see vol. i. p. 496). *Double hemiplegia* (also known as *pseudo-bulbar paralysis*) describes a variety in which there is evidence of paralysis on each side of the body, the result of a lesion on each side of the cerebrum, or it may be of the pons, and the alternative name is used because of the close resemblance in the clinical characteristics of such cases to those of cases of degenerative affection of the nuclei of the bulb—cases of true bulbar paralysis. There is also a condition known as *hysterical* or *functional* hemiplegia, but the qualifying adjective does not imply any marked difference in the characters of the paralysis so much as in its originating conditions. The variety known as *infantile* hemiplegia, so far as the paralysis both in its extent and distribution is concerned, does not differ in any important essential from the adult type, but does differ in the time of onset, probably also in the nature of the lesion underlying it, and in having certain other symptoms associated with the paralysis.

In this article it is proposed to consider the condition of hemiplegia as it manifests itself clinically in an adult—(1) at the onset of the affection, (2) when the paralysis has become well defined in all its symptoms; and also the condition as it occurs in children—the so-called infantile hemiplegia just alluded to—differing in certain important particulars from the condition in the adult.

(i.) *The Condition in a case of Hemiplegia at its onset.*—When the patient is seen the history of the mode of onset is of importance, not only as to the nature of the lesion but also in reference to prognosis and treatment. Frequently the account given is that the paralysis has occurred during sleep, although this is often difficult to verify. Not uncommonly the weakness has only become obvious when the patient gets out of bed, not aware of the paralysis, and then the history obtained is to the effect that the patient fell down and at once became paralysed, the true sequence of events being that the patient was already paralysed and fell down in consequence of the unilateral weakness. Such a patient when first seen is quite conscious and is paralysed on one or other side. The arm in the great majority of cases is more affected than the leg, and hangs usually limp and useless by the patient's side. In a case with such a history there is frequently no rigidity, and the deep reflexes may be normal at first or even diminished. In the paralysed limbs it is found that the most highly-developed movements are those that have suffered most. Thus the fingers may be completely motionless while fair power can be exerted at the shoulder and elbow and even at the wrist. Occasionally it is found that the shoulder is much affected, and then the hand may not be completely paralysed, but even in such a case it is always found to be considerably impaired in its movements. In the lower limb the foot does not necessarily suffer most, and indeed the hip movements may be most interfered with.

In certain cases of hemiplegia, however, as already stated, the leg is more affected than the arm. This is due to a difference in the *position* of the lesion in the brain, not to any difference in its character,—the lesion being in such a case so situated as to affect more the leg centre in the cortex or the leg fibres in the white substance or in the internal capsule, than the corresponding structures for the upper limb. But on account of the fact that the leg is represented on each side of the brain—is bilaterally represented, to use the ordinary phrase—in a much greater degree than the arm, the paralysis

of the leg, even in such a case as that now referred to, is never so complete as is the paralysis of the arm in an ordinary case. Thus it is rare to find in a case of this kind complete abolition of all movement in the leg, while it is not at all uncommon to find in a case in which the upper limb has suffered most that the arm is all but completely paralysed.

Besides the affection of the limbs, that of the face may be obvious or may have to be carefully looked for. The lower part of the face suffers more than the upper, the reason being that the movements of the upper part of the face on the two sides are much more closely associated in their movements than those of the lower part of the face, and consequently they are represented in a correspondingly greater degree on the same side of the brain. Thus there may be, indeed there usually is, distinct failure of ability to raise the upper lip on the affected side in any attempt to show the teeth, while there may be no appreciable difference in the two sides when an attempt is made to wrinkle the forehead. The tongue is deflected to the paralysed side when protruded, the stronger muscles of the healthy side pushing it towards the weak side of the body.

The trunk muscles also suffer, but this is only seen, as a rule, on forced movement, the reason again being that the muscles of the one side are very closely associated in their habitual movements with those of the opposite side, and are consequently represented on each side of the brain, and therefore suffer comparatively little in a unilateral lesion. But a curious and important fact has been pointed out by Dr. Hughlings Jackson, viz., that in quiet respiration the movement of the paralysed side of the chest may be greater than that of the opposite side, while in voluntary respiration the side on which paralysis is present moves less than the other side.

Such, then, are the chief motor symptoms which are found in a case of this nature as it comes before the observer in an early stage, and the lesion causing such a condition may be situated in the cortex, underneath this, or in the internal capsule, crus, or pons. There may, however, be other conditions present. Thus marked impairment of sensation may coexist with the unilateral motor impairment—a condition pointing almost invariably to a lesion in the posterior part of the internal capsule where the sensory fibres from the whole of the opposite side of the body are transmitted to their still somewhat obscure termination in the cerebrum. In such a condition it is not unusual to find the leg more affected than the arm, because of the closer proximity in this part of the fibres subserving the leg movements to those subserving sensation. For a similar reason, when sensation is found to be impaired, the visual condition should be carefully examined, for as the fibres of the optic radiation run very near the spot at which such a lesion would exist, hemianopsia as a result of damage to them may, and frequently does, coexist with sensory impairment.

Such, then, is the usual condition as regards distribution and character of paralysis in a case of ordinary hemiplegia at the onset.

In a certain number of cases, however, the face, instead of being affected on the same side as the body, is paralysed on the opposite side. This is due to the fact that the lesion causing the paralysis is so situated as to affect the facial fibres *below* the nucleus, and therefore on the same side as that of the face to which they are distributed. Such a lesion, however, being situated *above* the decussation of the pyramids, will naturally cause paralysis of the limbs and body of the opposite side. This position of the lesion accounts not only for this so-called "crossed" paralysis, but it also accounts for certain features in the characters of the facial paralysis and for some associated paralysis. Thus, in such a lesion the upper part of the face is more affected than in cases of ordinary hemiplegia—the paralysis approaching closely in type to the ordinary type of facial paralysis resulting from an affection of the facial nerve (*see* "Facial Nerve, Paralysis of"). Then, also, in such cases there is usually associated with the facial paralysis an affection of the sixth nerve, causing paralysis of the external rectus muscle of the eye on the same side. The close proximity of the sixth nerve nucleus to the facial in the pons will explain this as well as an occasional involvement of the sensation of the face—from affection of the fifth nerve. Another form of crossed hemiplegia is also met with in which the functions of the third nerve are interfered with on one side and of the face and of the limbs on the opposite side. Such a condition can only result from a lesion—if there be a single lesion—in the *crus cerebri*, where the motor tract for the opposite side of the body and the third nerve of the same side are in close apposition.

A lesion also just above the decussation of the pyramids affecting the hypoglossal nerve for one side and the motor tracts of the other, may cause paralysis of the limbs on one side, and of the tongue on the opposite side, without any associated affection of the face. Such a lesion, however, is rare, and would almost certainly prove fatal on account of its proximity to vital structures in the bulb.

Reference has been made only so far to a condition of hemiplegia, commencing with little if any impairment of consciousness. Such a condition is usually the result of blocking of a small artery, although at first the dynamic effect of such a lesion may extend much beyond the area supplied by the occluded vessel, and so the initial paralysis be much more severe and

extensive than it is ultimately. There may, however, in another class of cases be grave interference with consciousness at the onset, or even profound coma; and although all that has been said as to the character and distribution of the paralysis in a case of hemiplegia coming on without initial loss of consciousness is equally true of a case in which consciousness is lost at the onset, after consciousness has been restored, something must be said as to the initial condition in such a case, as it not infrequently renders the diagnosis considerably more difficult. The three most common causes of hemiplegia are—blocking of vessels by thrombosis, blocking by embolism, or the rupture of vessels. The conditions associated with the onset of the first have already been alluded to. Hemiplegia resulting from embolism is sudden in its onset. It may be unattended by loss of consciousness, and may very quickly pass off; or it may result in profound loss of consciousness lasting for hours at least, and convulsions may occur at the onset and be repeated during the unconsciousness. The character of the paralysis may be difficult to recognise during the unconsciousness. The whole condition of the patient may be one of apparently flaccid paralysis, but if careful watch is kept, the occasional voluntary movement of a limb on one side and the complete absence of any movement in those of the opposite side, may indicate the nature of the illness. Occasionally also there is considerable restlessness on the part of the patient, especially in cases of septic emboli, and on careful observation the absence of movement on one side may be detected. The knee-jerks may be completely lost, they will probably be so if the coma is profound, and deglutition may be interfered with as well as the action of the bladder and rectum. After the unconsciousness has passed of—although it must be remembered that it does not always do so—the condition of paralysis will be such as has already been described, and the position of the lesion will be determined from a consideration of the facts already mentioned.

A condition of cerebral hæmorrhage causing hemiplegia may have been preceded for some time by headache. On the other hand, it may occur suddenly when a patient is feeling particularly well. Consciousness may be lost at once, and a condition of profound coma supervene. Or the loss of consciousness may be slow, taking a few minutes or even, in the so-called "*ingravescent apoplexy*," a few hours. When consciousness is lost at once, and profound coma comes on, the hæmorrhage is probably into the ventricle, and it may be difficult to detect the signs of the unilaterality of the lesion. If consciousness be slowly lost, the unilateral weakness will probably be distinct before the onset of unconsciousness, and when this comes on the one-sided nature of the paralysis may become somewhat masked. During unconsciousness marked deviation of the eyes may be observed, there is usually difficulty in swallowing, and there may be retention or incontinence of urine, and loss of control over the rectum. The skin usually assumes a greasy moistness, and trophic changes over points of pressure may occur rapidly. The unconsciousness may deepen to death, or there may be recovery, consciousness becoming restored after several days. When this happens there is not infrequently intense headache which is distressing and often intractable. The condition of paralysis is then recognisable, and its characters answer to those already described.

The *prognosis* in any case of hemiplegia in the early stages is beset with difficulties. In cases of embolic hemiplegia, if not very severe and occurring in young adults, it is often possible to predict a speedy recovery. But it must always be remembered that the condition causing the embolism if it still persist may give rise to a second one, sooner or later. The writer has known a patient three days after an attack of embolic hemiplegia from which he had recovered, have another attack from which recovery was only partial. And in many cases the recovery is but slight, and a condition of considerable paralysis remains permanently. Of cases of hæmorrhagic hemiplegia the prognosis as regards life is good after the first fortnight, and a certain degree of recovery can always be predicted. In the first fortnight, however, the patient is in imminent danger of death, either as a result of changes occurring around the first hæmorrhage, or because of the occurrence of a second. And as the vascular and other conditions underlying the hæmorrhage still persist, a second hæmorrhage in the near future is almost inevitable. In cases of thrombosis, due to senile atheroma, there is not, as a rule, an extension of the blocking, but recovery, partial it is true, but often considerable, takes place. The recovery in the first few days is often marked, probably, however, because much of the paralysis at first was dynamic in origin and not the result of actual structural change. After that recovery is slower, and is never complete. If the thrombosis occur in vessels, the seat of syphilitic endarteritis, or from blood states, such as are met with during pregnancy or the puerperium, complete recovery is not to be looked for. If the paralysis be the result of tumour or abscess, the degree of recovery depends upon the removability of the abscess or tumour by surgical or other means.

The pathological conditions underlying the paralysis and the differential diagnosis and treatment of these different conditions will be found described in the diseases of brain vessels (see "Brain," vol. i.).

(ii.) It will now be well to consider the condition of hemiplegia as it is met with in a

patient some months after the onset of the condition. Whatever may have been the nature of the initial lesion the subsequent condition is the same.

Motor Symptoms.—There is, in the ordinary case, weakness of the whole of one side of the body—the face, arm, and leg being most obviously affected, although the trunk muscles also suffer. It may just be mentioned here that although the more distinct weakness is of one side, the other side is also affected, but in a much slighter and less noticeable way. The lower part of the face suffers most, and there is an obliteration of the naso-labial fold and distinct drooping of the upper lip on an attempt at showing the teeth. The tongue is protruded towards the paralysed side. The arm is much less helpless than at the onset of the paralysis, but there is more movement at the shoulder and elbow than at the wrist or in the hand, although this is sometimes obscured by the presence of adhesions at the shoulder, considerably limiting movement there. The whole arm is stiff, or, to use the ordinary term, rigid, but this rigidity can at first be overcome, although later, through contractions taking place, structural alterations at the joints and in the muscles make even passive movements restricted and difficult. There is in the ordinary case much more affection of the finger movements than of any others. The leg is similarly affected, although in less degree. The rigidity, however, is distinct. In some cases of hemiplegia, as has already been said, the leg is more affected than the arm, and in such a case there is inability to walk for a long time even after a fair degree of power is present in the arm and even in the hand; yet in such a case, no doubt because of the bilateral representation of leg movement in the brain, recovery in the leg usually proceeds to such a stage as to permit walking.

The reflexes may be alluded to. In the leg there is usually ankle clonus and the knee-jerk is exaggerated, and there may actually be a knee clonus. This exaggeration, although more marked on the paralysed side, is present also on the other, and even ankle clonus occasionally occurs on the so-called healthy side. In the arm there is exaggeration of the jerks elicited on tapping the wrist and elbow, and attention has recently been called by Babinsky and others to the fact that stroking the sole of the foot, in this and other conditions in which the lateral columns in the cord are degenerated, elicits a movement of extension of the big toe, the normal one being one of flexion. Sometimes by depressing the lower jaw and tapping the chin, a well-marked jaw-jerk may be obtained.

Occasional irregular movements are present on the affected side in hemiplegia. The most common of these is athetosis, in which the hand on the affected side undergoes a cycle of slow involuntary movements. The movement may also affect the arm and shoulder, so that the arm is elevated above the head in a grotesque and striking manner. Similar movement may be present in the foot and at the ankle, rarely at the other parts of the leg, and not infrequently about the face and neck, the platysma being apparently the muscle most implicated. Marked tremor may also be present on the paralysed side, resembling that of disseminated sclerosis in being evoked only on movement, but both these forms of movement are much more common in the cases of hemiplegia occurring in early life, so-called *infantile hemiplegia*, to which reference will presently be made.

Besides these evidences of motor paralysis sensory impairment may also be present. This, as we have already said, is more likely to be met with in cases in which the leg is more affected, and consists in a blunting to all forms of sensory stimulation. The special senses may also be involved. Hemianopsia, *i.e.* abolition of function of the half of each retina on the side of the lesion, resulting in blindness of the half of each visual field on the paralysed side, is the form in which vision is affected. Hearing, taste, and smell may be impaired on the paralysed side.

This is probably the most suitable place in which to refer to the speech and articulatory defects in hemiplegia, because they are partly sensory and partly motor. In nearly every case even of *left* hemiplegia there is at first some difficulty with articulation resulting in a slurring or blurring of what are usually clearly articulated definite sounds. This may pass off in a few days, or weeks, but in a certain proportion of the cases persists throughout. In the majority of cases of *right* hemiplegia, however, speech proper is affected. The motor processes of speech may be interfered with, so that the patient is unable to fit words to things or to ideas, resulting in the condition known as *motor* aphasia. The patient knows what he wants to say, but cannot say it, yet is able to recognise it when it is said, and to understand anything spoken to him; or he may be unable to fit written characters to heard words, a condition known as *agraphia*. Another defect, however, may be present, so-called *sensory* aphasia, in which the patient is unable to understand what is said to him,—so-called auditory aphasia; or is unable to interpret seen objects, *e.g.* unable to read—so-called visual aphasia or word-blindness. All these defects only occur in cases of right hemiplegia, or in cases of left hemiplegia in a left-handed person. The writer has seen a case, however, of left hemiplegia with aphasia in a right-handed woman. This patient's father, curiously enough, was left-handed in a marked degree. And it should be remembered that all cases of right hemiplegia do not suffer from aphasia, and that those most likely to escape are for obvious

reasons cases in which the leg suffers more than the arm (*see* "Aphasia," vol. i.).

Trophic and other Changes.—Besides the motor and sensory symptoms already described, certain changes, depending probably upon some change in the nutrition of the affected side, are usually present. Sometimes the limbs are colder and more blue, as if there were some venous engorgement due to vaso-motor paralysis. Frequently there is a marked diminution in the size of the limbs as compared with those of the opposite side, the result in some degree, no doubt, of the absence of physiological exercise, in some degree, perhaps, of the actual cerebral lesion and its effect on nutrition. Often the diminution in the limb is associated with changes in the joints, especially the shoulder-joint, which may become fixed and immobile. When this is present the diminution in the size of the upper limb may be very striking.

A few words are also necessary in reference to mental changes which may be present. In the ordinary case these are not very marked, usually consisting in a certain degree of emotional instability, so that the patient easily laughs or cries, and in a tendency to be easily irritated. Occasionally in the early stage actual acute maniacal symptoms may supervene, but this is at least rare in the later stages, if it occurs at all.

In some cases convulsions occur at intervals after the paralysis is past. These may be unilateral in distribution, or bilateral. They probably are to be expected in cases in which the lesion is just underneath the cortex.

Actual neuritis may occur in a paralysed limb, characterised by great pain, glossy skin, and changed electrical reactions. Such a complication the writer has seen most frequently in people of a gouty constitution, especially if glycosuria happened to be present. It is nearly alway associated with joint changes, especially in the shoulder-joint.

Treatment.—In the acute condition, at the time of onset, this must depend upon the diagnosis. If the hemiplegia is associated with a feeble pulse, if it has come on without loss of consciousness in an old person, the condition is probably one of thrombosis, and rest, quiet, easily assimilated food, and the somewhat free use of strychnia and alcohol, are the measures most likely to be successful. If the patient is younger and the condition is regarded as one due to syphilitic arteritis, then the usual anti-syphilitic remedies must be energetically used. But when once thrombosis has occurred, and necrotic changes have taken place in the area subserved by the blocked vessel, even the most energetic treatment is not likely to be successful in preventing permanent paralysis. If the case be one of embolism, rest is essential. The cardiac condition must be carefully watched, and cardiac tonics used if necessary. If, however, unconsciousness be present, and the patient have a tense pulse and a hypertrophied heart, then probably intracranial hæmorrhage is the condition present, and free purgation and other means of lowering tension and ridding the blood of poisonous material are the beginning and the end of effective therapeutics. Should the hemiplegia be the result of tumour, abscess, or fracture, these conditions must be dealt with *secundum artem.*

As regards the condition when it has become chronic, not much in the way of treatment will influence it. Attention is to be chiefly directed to maintaining good nutrition and preventing contractions and other deformities. Contracture is nearly always flexor, and attention should be directed to securing adequate stimulation of the extensor muscles by means of electrical treatment or resistance exercises. Massage and passive movement are also useful in preventing or in minimising contractures.

The pathological changes underlying the condition described have already been dealt with in considering the results of disease of the brain vessels (*see* vol. i. p. 475).

(iii.) *Infantile Hemiplegia.*—Although the state of a patient suffering from this form of paralysis, after the condition is well established, differs in no important particular from that first described, there are certain additional symptoms, especially associated with its onset, which mark it out as a distinct variety, worthy of special mention.

Infantile hemiplegia is the term applied to that form of hemiplegia which occurs in early life, usually in the first six years of life. It is ushered in, as a rule, with general malaise, with high fever and one severe unilateral convulsion or a series of such. The convulsion may spread so as to affect both sides. The condition of the child at this stage is one of grave and not infrequently fatal illness. The convulsive attacks may persist during several days, and when they cease the child is found to be paralysed on one side. At first the paralysis is flaccid, usually without sensory impairment, although hemianopsia is said to be frequently present. If the paralysis is right-sided, and the child had been able to speak, speech may be much interfered with, but only temporarily, the child probably regaining the power of expression by the education of the corresponding centres of the other hemisphere.

If a patient who has suffered in this way is seen a few years after the attack, the condition resembles closely that already described as occurring in the adult. There is the same spastic rigidity with contracture affecting the arm more than the leg, the same exaggeration of reflexes, and a similar but much more marked difference in size and development between the limbs on opposite sides, those on the paralysed side, especially the arm, being much smaller.

The involuntary movements already alluded to, especially athetosis, are met with characteristically in this condition, and unilateral convulsions are of frequent occurrence. These may remain absent even for years after the initial convulsions, but they are apt to recur. In some cases the convulsions are severe, in others the attacks are more those of *petit mal*, and they may be associated with post-epileptic automatism. The mental condition is nearly always impaired, and not infrequently a condition of imbecility is present which renders the child suitable only for an asylum or a similar institution.

Pathology and Morbid Anatomy.—The condition is probably an inflammatory one of the cortex. Whether this is primarily in the cells or the result of vascular blocking, is not yet certain, but it seems not unlikely that an organised virus is the primary cause of whatever results in vessels or brain cells. When a child recovers from the initial grave condition and lives for years afterwards, the brain is then found to be in a cystic condition (porencephaly) with degeneration around it, a condition which, it is obvious, cannot inform us as to the primary cause of the brain lesion.

Prognosis and Treatment.—At the commencement of the illness life is gravely threatened, and death can only be prevented by careful and judicious treatment. Tepid sponging may do much to reduce the temperature; bromide by the rectum may control the convulsions, and should be given freely. A dose of calomel should be administered as soon as the condition is recognised, and care exercised that the child gets sufficient nourishment of a light, easily-digested quality.

If the acute condition be recovered from the prognosis is grave as regards anything like complete recovery of power, and the possibility of great physical impairment, of the frequent occurrence of convulsions, and of much mental change, must be borne in mind in forecasting the future. Not much can be done by drugs after the hemiplegia is established. The convulsions, however, can usually be completely controlled by the judicious use of bromide combined with arsenic and nux vomica, and by carefully chosen exercises and passive movements physical development may be aided and deformities prevented.

If the mental condition be impaired just after the onset there is not much chance that it will ever return to normal, and as soon as possible the child should be sent to an institution.

Hémiplégie Flasque.—The name given by Bouchard to cases of cerebral hæmorrhage, occurring most commonly in children, in which contracture and rigidity do not occur as late symptoms. *See* PARALYSIS (*Infantile Hemiplegia, Prognosis*).

"Hemisine."—Epinephrine or "Hemisine" (B. W. & Co.) is stated to be the active principle of the medulla of the adrenal gland; it increases the rapidity and strength of the heart's action and causes constriction of the smaller arteries; it blanches a mucous membrane when applied to it; it has been usefully employed in hæmorrhages except those of cerebral or pulmonary origin, has been combined with cocaine or eucaine in the production of local anæsthesia, and has been used to diminish the congestion of inflamed mucous membranes (*e.g.* in conjunctivitis, hay fever, etc.); the dose varies from $\frac{1}{200}$ grain to $\frac{1}{64}$ grain. *See* ADRENAL GLANDS, ADRENALIN.

Hemiterata.—Anomalies of formation of a minor degree as distinguished from *terata* or monstrosities proper, and due to a less powerful teratogenic cause acting in a localised fashion.

Hemlock. *See* CONIUM; TOXICOLOGY (*Alkaloids and Vegetable Poisons, Spotted Hemlock*).

Hemp, Indian. *See* CANNABIS INDICA; TOXICOLOGY (*Indian Hemp*).

Henbane. *See* HYOSCYAMUS; TOXICOLOGY (*Alkaloids and Vegetable Poisons, Henbane*).

Hendon Epidemic.—An epidemic of scarlet fever traced to the milk taken from cows suffering from a disease analogous to scarlet fever (characterised by loss of hair and by a vesicular eruption on the teats and udders).

Henle's Loop. *See* PHYSIOLOGY; EXCRETION (*Secretion of Urine, Structure of the Kidney*).

Henoch's Purpura. *See* PURPURA (*Varieties, Henoch's*).

Henpuye.—Dog-nose or goundu, a disease frequently seen in the natives of the Gold Coast, consisting of a bilateral swelling of the sides of the nose, osseous in character, and causing much deformity. *See* NOSE, CHRONIC INFECTIVE DISEASES (*Henpuye*).

Hepata Succenturiata.—Accessory hepatic lobes, an occasional anomaly of the liver. *See* LIVER, DISEASES OF (*Tongue-like Lobes*).

Hepatalgia.—Pain in the liver, due to abscess, gall-stones, cancer, inflammation, etc. *See also* HYSTERIA (*Sensory Disorders*).

Hepatectomy.—Resection of the liver for the removal of growths (*e.g.* tuberculomata, gummata, etc.) or of Riedel's lobe.

Hepatic.—Relating to or affecting the liver (Gr. ἧπαρ, the liver); for *hepatic abscess, see* Liver, Tropical Affections of (*Perihepatitis, Suppuration*); for *hepatic aneurysm, see* Hæmatemesis (*Causes*); for *hepatic colic, see* Colic (*Diagnosis from other Colics*); and for *hepatic diseases, see* Liver, Diseases of.

Hepaticostomy.—The making of an artificial opening into the hepatic duct.

Hepatisation.—The consolidation of the lung tissue so that it comes to resemble that of the liver, met with in pneumonia, and described as red (in the second stage of pneumonia), grey and yellow (in the third stage), or white (in syphilis). *See* Pneumonia (*Physical Signs*).

Hepatitis. *See* Gall-Bladder and Bile-Ducts, Diseases of (*Cholangitis*); Liver (*Acute Hepatitis, Tropical Abscess*).

Hepatocele. *See* Liver, Diseases of (*Displacements, Hernia*).

Hepatocholangioenterostomy.—The establishment of a direct communication between the parenchyma of the liver and the intestinal canal (*Kehr*).

Hepatolith.—A biliary calculus. *See* Gall-Bladder, Diseases of (*Gall-Stones*).

Hepatomphalos.—Congenital herniation of the liver at the umbilicus. *See* Liver, Diseases of (*Displacements*).

Hepatopexy.—Fixation of the liver in its normal position by surgical means (*e.g.* suturing it to the anterior abdominal wall or costal arch). *See* Liver, Diseases of (*Hepatoptosis*).

Hepatoptosis.—Wandering or floating liver, prolapse of the liver. *See* Enteroptosis; Liver, Diseases of (*Hepatoptosis*).

Hepatorrhexis.—Rupture of the liver. *See* Abdomen, Injuries of (*Contusion, Lesions of the Liver*).

Hepatotomy.—Incision of the liver.

Hephæstic Hemiplegia.—Hammerman's cramp (Gr. Ἥφαιστος, Vulcan, master of the arts of working in metal, etc.) *See* Neuroses, Occupation.

Herapathite.—The iodo-sulphate of quinine (named after the chemist, Herapath), and formerly used in scrofula.

Herculesbad. *See* Balneology (*Austria, Hungary*).

Herculeus Morbus.—Epilepsy (*q.v.*).

Hereditary Ataxia. *See* Paralysis (*Hereditary Ataxia, Friedreich's Disease*).

Heredity.

See also Alcoholism (*Etiology, Heredity as a Factor*); Amblyopia (*Toxic, Hereditary Neuritis*); Asthma (*Nature and Etiology*); Chorea (*Hereditary Adult Chorea*); Colour Vision (*Influence of Heredity*); Convulsions, Infantile (*Etiology*); Deafmutism; Diabetes Insipidus (*Etiology*); Gout (*Heredity*); Hæmophilia (*Etiology*); Hay Fever (*Etiology, Heredity*); Heart, Affections of Myocardium and Endocardium (*Etiology*); Hysteria (*Etiology*); Insanity, Etiology of (*Heredity and Neuroses*); Life Insurance (*Family History*); Lung, Tuberculosis of (*Etiology, Heredity*); Maternal Impressions; Nephritis (*Etiology, Renal Cirrhosis*); Obesity (*Etiology*); Paralysis (*Primary Lateral Sclerosis, Causation*); Paralysis (*Hereditary Ataxia*); Pregnancy, Multiple (*Predisposing Influences*); Senile Insanity (*Pathogenesis*); Spasm (*Paramyoclonus multiplex*); Syphilis (*Syphilis in Children, Hereditary*); Tetany (*Causation*); Typhoid Fever (*Etiology, Family Predisposition or Immunity*).

Heredity is a term for the relation of organic or genetic continuity which binds generation to generation. It is an anachronism to speak of it as a power, or principle, or force. Similarly, inheritance may be defined as all that the organism is or has to start with in virtue of its genetic relation to its parents and ancestors. The central problem of heredity is to arrive at an accurate conception of the genetic relation between successive generations; the central problem of inheritance is to measure the resemblances and differences in the hereditary characters of successive generations, and to find, if possible, some general formula which will sum up the facts.

Physical Basis of Inheritance.—If we mean by inheritance all that an organism is or has to start with in virtue of its genetic relation to its parents and ancestors, then it is plain that the physical basis is in the fertilised ovum. There is, as regards property, an obvious distinction between the inheritance and the person who inherits, but no such distinction is possible in biology, for the fertilised ovum *is* the inherit-

ance and is at the same time the potential inheritor. This is a biological commonplace, as a statement of fact quite indisputable, nevertheless bristling with difficulties. Some of these difficulties, however, are incidental, not intrinsic. Thus, though it is interesting to ask how a heritable organisation, supposed to be very complex, may be imagined to find physical basis in a microscopic ovum and in a spermatozoon which may be only $\frac{1}{100000}$ of the ovum's size, the same sort of question may be raised in regard to ganglion cells; it is not distinctively a problem of heredity. It may, however, be recalled (1) that the physicists report that the image of a Great Eastern filled with framework as intricate as that of the daintiest watch does not exaggerate the possibilities of molecular complexity in a spermatozoon, whose actual size may be less than the smallest dot on the watch's face; (2) that in development one step conditions the next, and one structure often grows out of another, so that we are not forced to stock the microscopic germ-cells with more than initiatives; and (3) that development implies an interaction between the growing organism and a complex environment, without the stimulus of which the inheritance would remain unexpressed, and that the full-grown organism includes much that was not inherited at all, but has been acquired as the result of nurture or external influence. And, again, it is altogether inexpedient to lay on the shoulders of the student of heredity the burden of interpreting the orderly and correlated succession of events by which the fertilised egg-cell gives rise to an embryo. This is the unsolved problem of physiological embryology, and raises questions quite distinct from those of heredity and inheritance.

But when these incidental difficulties are set aside as irrelevant, there remains the intrinsic difficulty of accounting for the germ-cell's complex, ready-made organisation and marvellous potentiality. One suggestion is expressed in the theory of pangenesis, which occurred at intervals in the long period between Democritus and Darwin. On this theory the cells of the body are supposed to give off characteristic and representative gemmules; these are supposed to find their way to the reproductive elements, which thus come to contain representative samples of the various components of the body, and are therefore able to develop into an offspring like the parent. This theory involves many hypotheses, and is avowedly unverifiable in direct experience, but it is more to the point to notice that there is another theory of heredity which is on the whole simpler, which does, on the whole, fit the facts better.

This second theory is expressed in the phrase "germinal continuity," and has been independently expressed by a number of biologists, though Weismann has the credit of its elaboration. There is a sense, Mr. Galton says, in which the child is as old as the parent, for when the parent's body is developing from the fertilised ovum, a residue of unaltered germinal material is kept apart to form the future reproductive cells, one of which may become the starting-point of a child. In many cases scattered through the animal kingdom (*e.g.*, *Ascaris* and *Sagitta* among worms, *Moina* among crustaceans, *Chironomus* among insects, Phalangidæ among arachnids, *Micrometrus aggregatus* among fishes) the beginning of the lineage of germ-cells is demonstrable in very early stages before the differentiation of the body-cells has more than begun. Thus in the development of *Ascaris megalocephala* of the horse, according to Boveri, the very first cleavage divides the fertilised ovum into a cell which is the ancestor of all the somatic cells, and another, which is the ancestor of all the germ-cells.

But in many other cases, notably in plants and in the higher animals, the segregation of germ-cells is not demonstrable until a relatively late stage. Therefore, while the keystone of Weismann's theory is that the germinal material which starts an offspring owes its virtue to being materially continuous with the germinal material from which the parent or parents arose, he does not suppose a continuous lineage of recognisable germ-*cells* (for this is often unrecognisable), but a continuity of the germ-*plasm*; that is, of a specific substance of definite chemical and molecular structure which is the bearer of the hereditary qualities. According to Weismann, a part of the germ-plasm contained in the parent egg-cell is not used up in the construction of the body of the offspring, but is reserved unchanged for the formation of the germ-cells of the following generation. Thus the parent is rather the trustee of the germ-plasm than the producer of the child; and in a new sense the child is a chip of the old block. Similar material to start with, similar conditions in which to develop, *therefore*, like tends to beget like.

It should be carefully noticed that while early segregation of the germ-cells is in many cases an observable fact—and doubtless the list of such cases will be added to—the conception of the "germ-plasm" is hypothetical, just as the conception of *a* specific living stuff or "protoplasm" is hypothetical. In the complex microcosm of the cell we cannot point to any one stuff and say "this is protoplasm"; and it may well be that vital activity depends upon the interactions of several complex stuffs which, like the members of a carefully-constituted form, are characteristically powerful only in virtue of their interrelations. Still less can we demonstrate the "germ-plasm," even if we were able to show that its physical basis is in the chromosomes of the nucleus. The theory has to be judged, like all conceptual formulæ, by its adequacy in fitting facts.

Dual Nature of Inheritance.—It is a familiar fact that, apart from exceptional cases (*e.g.* asexual multiplication and parthenogenesis), the inheritance of a multicellular organism is dual, part of it coming from the mother and part of it from the father. The more we know in regard to fertilisation, the clearer does the general fact become that there is an intimate and orderly union of maternal and paternal contributions. Professor E. B. Wilson sums up the present state of opinion somewhat as follows:—As the ovum is much the larger, it is believed to furnish the initial capital—including it may be a legacy of food-yolk—for the early development of the embryo. From both parents alike comes the inherited organisation which has its seat (according to many) in the chromosomes of the nuclei of ovum and spermatozoon. From the father comes the centrosome which organises the machinery of cleavage and distributes the dual inheritance equally between the daughter-cells. Recent discoveries confirm Huxley's prophecy (1878):—"It is conceivable, and indeed probable, that every part of the adult contains molecules derived both from the male and from the female parent; and that, regarded as a mass of molecules, the entire organism may be compared to a web of which the warp is derived from the female and the woof from the male." "What has since been gained," Wilson says, "is the knowledge that this web is to be sought in the chromatic substance of the nuclei, and that the centrosome is the weaver at the loom." Four saving clauses seem necessary at this stage in our discussion:—(1) What we have called the second great fact of inheritance does not imply that the dual nature of the inheritance must be *patent* in the full-grown offspring, for hereditary resemblance is often markedly unilateral. (2) Though inheritance is immediately dual, it is in quite as real a sense multiple, from ancestors through parents. (3) If Loeb is able to induce artificial parthenogenesis in sea-urchins' eggs exposed for a couple of hours to sea-water to which some magnesium chloride has been added; if Delage is able to fertilise and to rear normal larvæ from *non*-nucleated ovum-fragments of sea-urchin, worm (*Lanice*), and mollusc (*Dentalium*), we should be chary of accepting too readily the conclusion that the nuclei are the exclusive bearers of the hereditary qualities. (4) The fact that an ovum without any sperm-nucleus, and an ovum-fragment without any but a sperm-nucleus, can in some cases develop into a normal larva, points to the conclusion, which other facts also suggest, that each germ-cell, whether ovum or spermatozoon, bears a complete equipment of hereditary qualities.

Different Degrees of Hereditary Resemblance.—The big treatise of Prosper Lucas (1847) may be said to close the period of *proving* hereditary resemblance. It is now legitimately taken for granted that the present is the child of the past, and that the past is represented in the child. An organism's start in life is vigorously determined by its parents and ancestors; not only specific characters, but trivial idiosyncrasies—not only physical qualities, but mental as well—not only the normal, but the abnormal may be transmitted. At the same time, it should be noted that this department of the study of heredity is by no means closed; thus some morbid conditions are much more likely to be transmitted than others, and we ought to have statistical estimates of the probabilities of transmission in each case. And, again, there are some subtle qualities whose heritability must not be assumed without evidence. Thus it is of great importance that Karl Pearson has recently supplied, for certain cases, definite proof of the heritability of fecundity, fertility, and longevity.

The large fact of inheritance which confronts us is the sensible stability of type from generation to generation. It is summed up in the familiar saying, "Like begets like." We know, however, that this saying is insufficient, since variation is as striking a fact as complete hereditary resemblance. A variation may imply some incompleteness in the offspring's re-expression of the parents' hereditary qualities, or it may imply the appearance of something new,—some novel molecular arrangement in the germ-plasm. In any case it leads us to modify the familiar saying, and to state more cautiously that like *tends* to beget like. But this platitude does not sum up even our familiar experience, and thus we are led to consider the different degrees of hereditary resemblance, for which a confused classification and a troublesome terminology have been suggested. The three most important cases seem to be blended, exclusive, and particulate inheritance. (*a*) In *blended* inheritance, the characters of the two parents, *e.g.* in regard to a particular feature, such as the colour of the hair, are ultimately combined in the offspring. This is particularly well seen in some hybrids, and is probably the most frequent mode of inheritance. (*b*) In *exclusive* inheritance, the expression of maternal or of paternal characters in relation to a given feature, such as eye colour, is suppressed. The resemblance is unilateral, and often crossed, the son taking after the mother and the daughter after the father. (*c*) In *particulate* inheritance there is in the expression of a given character a part which is wholly paternal and a part which is wholly maternal. Thus an English sheep-dog may have a paternal eye on one side, and a maternal eye on the other. Suppose the parents of a foal to be markedly light and dark; if the foal is light-brown or grey the inheritance is blended; if light or dark it is exclusive; if piebald, it is particulate in its mode of inheritance for that feature.

As already hinted, the different modes of inheritance are often well illustrated in hybrids between different species or breeds. The hybrid may be thoroughly intermediate between its

parents, the blending being more like the mingling of two pigments than the interweaving of warp and woof. Or it may show an exaggeration of the characters of one parent, often with little apparent realisation of the peculiarities of the other. These two cases correspond to blended and exclusive inheritance in ordinary mating within the same breed. But the hybrid may in other cases be very different from either parent, and exhibit features which appear to be novel, or seem interpretable as the reassertion of the characteristics of a remoter ancestor. In short, it may show either a new variation or a reversion. Perhaps the most extraordinary fact is that at least two of these different modes of inheritance may be illustrated in one brood or litter of hybrids.

From another point of view we may express the facts in terms of the quality of prepotency. It seems certain that in respect to certain characters the paternal inheritance is often more potent—more capable of finding expression—than the maternal, or *vice versa*; thus in man the father tends to be prepotent in the matter of stature, and breeders give many instances where certain, even trivial, characters of the sire or the dam reappear persistently in the offspring irrespective of the nature of the other parent. If, as Ewart and others maintain, this quality of prepotency tends to be developed by inbreeding, it may be frequent in nature, especially among gregarious and isolated groups, and it may explain the persistence of new variations in their incipient stages. It is interesting to note Reibmayr's thesis that the evolution of a successful human race implies alternating periods of dominant inbreeding and dominant cross-breeding. The former gives fixity to character, the latter averts degeneracy, and stimulates those new variations which form the raw material of progress.

Until more precise data accumulate in regard to blended, exclusive, and particulate inheritance, it will not be possible to simplify the matter with any security, but attention may be directed to Weismann's theoretical suggestion of a germinal struggle in the arcana of the germ-cells, a struggle in which the maternal and paternal contributions may blend and harmonise, or may neutralise one another, or in which one may conquer the other, or in which both may persist without combining.

Finally, in this connection, we must note that while it is a matter of observation that there are great differences in the degree in which offspring resemble their parents, it is a matter of conjecture that lack of resemblance must be due to incompleteness in the inheritance. Indeed, the fact that the resemblance so often reappears in the third generation makes it probable that the incompleteness is not in the inheritance, but simply in the expression of it. The characters which seem to be absent, to "skip a generation" as we say, are probably part of the inheritance all the time, but they remain latent, being neutralised, silenced (we can only use metaphors) by other characters, or unexpressed because of the absence of the appropriate stimulus. A neglect of this distinction is a frequent source of misunderstanding.

Filial Regression.—From generation to generation there is a tendency to keep up a specific average. "The large," Galton says, "do not always beget the large, nor the small the small; but yet the observed proportion between the large and the small, in each degree of size and in every quality, hardly varies from one generation to another." This is partly due to natural elimination, weeding out the extraordinary and the abnormal, often at or even before birth. But it is to be primarily accounted for by what Galton calls "filial regression." Karl Pearson gives a clear illustration:—take fathers of stature 72 inches, the mean height of their sons is 70·8,—a regression towards the mean of the general population; on the other hand, fathers with a mean height of 66 inches give a group of sons of mean height 68·3 inches,—again nearer the mean. "The father with a great excess of the character contributes sons with an excess, but a less excess of it; the father with a great defect of the character contributes sons with a defect, but less of it."

As Galton puts it, human society moves as a vast fraternity. The sustaining of the specific average is not due to each individual leaving his like behind him; it is due to a regression which tends to bring the offspring of extraordinary parents nearer the average of the stock. In other words, children tend to differ less from mediocrity than their parents. This big average fact is to be accounted for in terms of that genetic continuity which makes an inheritance not dual but multiple. A man is the product not only of his parents, but of his ancestry, and "unless very careful selection has taken place, the mean of that ancestry is probably not far from that of the general population." Pearson continues, "It is the heavy weight of this mediocre ancestry which causes the son of an exceptional father to regress towards the general population mean; it is the balance of this sturdy commonplaceness which enables the son of a degenerate father to escape the whole burden of the parental ill."

Law of Ancestral Inheritance.—Perhaps the most important general conclusion which has yet been reached in regard to inheritance is that formulated in Galton's law of ancestral inheritance, to which this authority was led by his studies on the inheritance of human faculties, and more particularly by a series of studies on Basset hounds. According to Galton's law, "the two parents between them contribute *on the average* one-half of each inherited faculty, each of them contributing one-quarter of it. The four grandparents contribute between them one-

quarter, or each of them one-sixteenth; and so on, the sum of the series $\frac{1}{2}+\frac{1}{4}+\frac{1}{8}+\frac{1}{16}+$ etc., being equal to 1, as it should be. It is a property of this infinite series that each term is equal to the sum of all those that follow, thus: $\frac{1}{2}=\frac{1}{4}+\frac{1}{8}+\frac{1}{16}+$ etc.; $\frac{1}{4}=\frac{1}{8}+\frac{1}{16}+$ etc., and so on. The prepotencies or subpotencies of particular ancestors, in any given pedigree, are eliminated by a law which deals only with *average* contributions, and the varying prepotencies of sex in respect to different qualities are presumably eliminated."

This law of ancestral inheritance, which states that each parent contributes on an average one-quarter, each grandparent one-sixteenth, and so on, must not be accepted as a dogmatic conclusion, but as an approximate statistical formula, and it must be noted that it applies most convincingly to cases of *blended* (not exclusive) inheritance. Pearson's paper on the "Law of Reversion" (1900) should be read as a supplement to Galton's *Natural Inheritance*. In connection with the number of ancestors and the mosaic or multiple nature of inheritance, it may be useful to recall that intermarriages greatly reduce the theoretical number of ancestors. Thus while Kaiser Wilhelm II. might have had 16, 32, 64, 128, 256, 512, 1024, 2048, 4096 ancestors in generations iv.-xii. respectively, he actually had 14, 24, 44, 74 in generations iv.-vii., and probably 116, 177, 256, 342, and 533 in generations viii.-xii.

Reversion.—This term may be conveniently used to include cases where, *through inheritance*, an individual exhibits some character or characters not expressed in the parents, but known to occur in ancestors. The character, normal or abnormal, whose reappearance is called a reversion, may be found within the verifiable family, within the breed, within the species, or even in a presumed ancestral species. Karl Pearson defines a *reversion* as "the full appearance in an individual of a character which is recorded to have occurred in a definite ancestor of the same race," while *atavism* is "a return of an individual to a character not typical of the race at all, but found in allied races supposed to be related to the evolutionary ancestry of the given race." But as the two words have been used by some others in the converse way, or as equivalent, and as it seems only a distinction of degree, the single term reversion may here suffice.

Good illustrations of reversion are furnished by hybrids. Thus, in one of Ewart's experiments a pure white fantail cock-pigeon, of old-established breed, which in colour had proved itself prepotent over a blue pouter, was mated with a cross previously made between an owl and an archangel, which was far more of an owl than an archangel. The result was a couple of fantail-owl-archangel crosses—one resembling the Shetland rock-pigeon, and the other the blue rock of India. Not only in colour, but in shape, attitude, and movements there was an almost complete reversion to the form which is believed to be ancestral to all the domestic pigeons. The only marked difference was a slight arching of the tail, which was, however, 12-feathered as usual, in contrast to the 30-feathered one in the fantail.

But great care is necessary in arguing from the results of hybridisation to those of ordinary mating, and even if some of the phenomena of exclusive inheritance seem to demonstrate reversion to a near ancestor, we need a broader basis of facts than we have at present before we can generalise. Karl Pearson (1900) has recently sought to formulate a general law of reversion supplementary to Galton's law of ancestral inheritance.

Many phenomena have been labelled reversions on the flimsiest evidence. Thus the occurrence of a Cyclopean human monster with a median eye has been called a reversion to the ascidian, and gout has been called a reversion to the reptilian condition of liver and kidneys. Often there is not the slightest attempt to eliminate the phenomena of arrested development or of abnormalities induced from without. Often, too, there has been no scruple in naming or even inventing the ancestor, to whom the reversion is supposed to point, although definite evidence of the pedigree is awanting; and the vicious circle is not unknown of arguing to the supposed ancestor from the supposed reversion, and then justifying the term reversion from its resemblance to the supposed ancestor. Little allowance has been made for coincidence, and the postulate of characters remaining latent for millions of years is made as glibly as if it were just as conceivable as a throwback to a great-grandfather.

Reversion is a phenomenon of exclusive inheritance, and the theoretical conception which it implies is that characters may be latent for a generation or for generations, or, in other words, that certain potentialities or initiatives which form part of the heritage may remain unexpressed for lack of the appropriate liberating stimulus, or for other reasons, or may have their normal expression disguised. There does not seem to be anything in this conception which is at variance with more securely established generalisations. But the danger is in applying the interpretation. Even when an individual exhibits the reappearance of an ancestral character which was not in his parents, this is not necessarily due to the reassertion of latent elements in the inheritance. It may be a case of ordinary regression; it may be a case of arrested development; it may be an individually acquired modification adventitiously induced, apart from inheritance, by a recurrence of suitable conditions of function or environment; it may be an extreme variation whose resemblance to an ancestral characteristic is a coincidence; and so

on. In short, what are called reversions are probably in many cases misinterpretations.

Transmission of Acquired Characters.—The question of the transmissibility of acquired characters has been much discussed within recent years, but there has not always been a clear apprehension of the point at issue. The individuals composing a species are neither quite like one another nor quite like their parents, and it is possible to measure these "*observed differences.*" As we come to analyse them, we discern that many structural peculiarities of the body can be shown by experiment to be definitely related to some alteration or peculiarity in environment or in function, that these are not even hinted at in the young forms, but begin to appear when the particular conditions begin to operate. These are called by biologists "*modifications*" or "*acquired characters,*" and it is in regard to these and these alone that the real argument has concerned itself. They may be defined as structural changes in the body of the organism induced by changes in the environment or in the function, and such that they transcend the limits of organic elasticity and therefore persist. Now when we eliminate from the total of observed differences of structure the somatic modifications which we have detected, there remain a number of differences which we call "*variations.*" These cannot be shown to result directly from functional or environmental stimuli operating upon the body; they are often hinted at even before birth; and they are not alike even among similar forms whose conditions of life seem absolutely uniform. Little that is certain is known in regard to their origin, but it is supposed that they result from changes in the germinal material before or in fertilisation; they are therefore called germinal, blastogenic, or congenital variations, and their transmissibility is indubitable. The precise question is, therefore, whether the modifications of the body can so specifically affect the reproductive cells that the next generation will inherit, in some measure at least, the modification acquired by the parent or parents. In other words, may the results of "nurture" be transmitted, or is it the "nature" alone that constitutes the inheritance?

Some of the frequent misunderstandings of the question must be referred to to clear the ground. (1) There is no relevancy in citing cases where a particular somatic change appears generation after generation, *e.g.* shortsightedness or gout, unless it is clearly shown that the change in question is really a "somatic modification," and not a congenital variation whose transmissibility is admitted by all. (2) It is a misunderstanding to cite cases where unicellular organisms, such as bacteria or monads, have been profoundly modified by culture, so that, for instance, the descendants of a virulent microbe are gradually led to lose their evil potency. This is irrelevant, because in regard to unicellular organisms we cannot draw the distinction between germinal matter and soma on which the definition of an acquired character depends. (3) There is little relevancy in citing cases where a particular bodily change appears generation after generation, unless it can be shown that the change reappears *in virtue of inheritance*, and not simply because the conditions of function and environment which evoked it in the first instance are still persisting to evoke it in those that follow. Reappearance is often confused with inheritance. (4) It is necessary to distinguish between the possible inheritance of a particular modification and the possible inheritance of indirect results of that modification. It is likely that some important modifications influence the general vigour of the body, and thus through nutrition the reproductive cells; but unless the offspring change *in the same direction* as that exhibited in the original parental modification, we are not warranted in speaking of the inheritance of an acquired character. (5) It is apt to be fallacious to appeal to data from not more than two generations. It has often been pointed out that mammals, such as sheep, taken to a new country, exhibit a change in the character and length of the hair, and that their progeny exhibit the modification in a still more marked degree. But unless statistics of the third generation at least are presented, such cases are of no value, for it is only natural that the second generation should show the modification in a more marked degree than their parents did, since the offspring were subjected to the modifying influence from birth, whereas their parents were influenced only from the date of their importation. (6) It is necessary to appreciate the distinction between a change of the reproductive cells *along with* the body, and a change of the reproductive cells *resulting from and representative of* a change in the body. Some poisoning of the system on the parent's part by alcohol, opium, or some virus, may be followed by degeneracy in the offspring. But if the fact be admitted, what is the correct interpretation? In some cases what is really inherited may be the degeneracy of nature which led the parent to, say, alcoholism, and which finds the same or another expression in the child. In other cases it may be that what looks like inheritance is the result of early infection before or soon after birth. In other cases it may be that there was in the parent a poisoning of the whole system — reproductive cells as well as body—which is not a case by which to test the transmissibility of an acquired character. (7) There is no doubt a sense in which every acquired character is congenital, for there must have been in the nature of the organism the rudimentary possibilities of it; and there is a sense in which every congenital character may be said to be acquired, since it

needs to be nurtured by appropriate conditions if it is to develop. And yet the distinction is not a verbalism. For although we do not suppose that the environment is creative, and although we must admit that the potentiality of the acquired character must be in the nature of the organism, just as the possibility of an explosion in the barrel of gunpowder, yet, as a matter of fact, it is possible to distinguish between the actual modification which we see and measure and the possibility of it which we presuppose. Similarly, while it is very true that the potentialities so marvellously embodied in the fertilised ovum require appropriate environing conditions if they are to be realised, for, as His observed long ago, "it is a piece of unscientific mysticism to suppose that heredity will build up an organism without mechanical means," yet this does not affect the validity of our definition of an acquired modification as distinguished from a congenital specific character.

The main argument against a belief in the transmissibility of acquired characters is simply that the evidence for the affirmative is extremely unsatisfactory. But this position is corroborated if we accept the view that the germ-plasm or the material basis of inheritance is in a marked degree apart from the general life of the body, and sometimes segregated at a very early stage in development. For this view raises a presumption against the likelihood of the germ-plasm being readily affected in a specific and representative manner by changes in the nature of the body cells. It must be allowed, however, that our inability to conceive of the mechanism by which an acquired character of the brain may affect the reproductive cells in a specific and representative manner does not prove that the supposed influence is an impossibility.

The argument in favour of the view that acquired characters may be transmitted is found in the large number of facts which may be readily interpreted on this hypothesis, and on a few facts which seem directly to suggest it. And the affirmative position is strengthened by a consideration of the unity of the organism. In many plants the distinction between somatic cells and germ-cells can hardly be drawn, and even if we keep to animals the bonds between the body and its germ-cells are often very close. The blood and lymph or other body fluids form a common medium for the various parts of the organism. Alteration of diet in the early youth of some animals, such as tadpoles, may determine the predominance of one sex or the other through influences which must pass from soma to germ-cells. Various poisons may affect the bodily system and the reproductive organs at the same time, and there are real though dimly understood correlations between the gonads and the rest of the system. It is therefore erroneous to think of the germ-plasm as if it led a charmed life uninfluenced by any of the accidents and incidents in the daily life of the body which bears it. No one believes this, Weismann least of all, for he finds one of the chief sources of congenital or blastogenic variation in the nutritive stimuli exerted on the germ-plasm by the varying state of the body. But it is one thing to admit that the germ-plasm has no charmed life nor insulation from bodily influences, and quite another thing to believe that a change in the body, induced by use or disuse or by change in surroundings, can influence the germ-plasm in such a definite way that the offspring will exhibit the same modification which the parent acquired, or even a tendency towards it.

Of the direct evidence suggestive of the transmissibility of acquired characters a few representative samples may be given. (1) The Panjabis are said to show peculiarities of musculature and skeleton, which are related to the frequency with which these people assume the squatting posture. But it may be that these modifications are acquired during each individual lifetime. The evidence is inconclusive, and we may set against it the case of the compressed foot of Chinese ladies, for there seems to be no evidence that the long-continued deformation has resulted in any hereditary change in the Chinese baby's foot. (2) The alleged dwindling of the little toe is instanced as a case of the inheritance of a modification induced by tight boots. But the evidence is flimsy; a dwindling has also been alleged in savages who do not wear boots; it is possible that there is in man, as there was in the horse-stock, a congenital variation in favour of a reduction of digits; and there are other possible interpretations. (3) In 1796 the utmost speed of the English trotter was a mile in 2 minutes 57 seconds; decade after decade the speed and the percentage of swift trotters increased; finally, there has been evolved a breed who can trot a mile in 2 minutes 10 seconds. This has been claimed as evidence of the cumulative transmission of the results of exercise or nurture. But this interpretation overlooks the results of selective breeding which may have increased the congenital swiftness, and the process of elimination which persistently weeded out the less swift from the stud. (4) In 1875 Schmankewitsch was able to transform one type of brine-shrimp, *Artemia salina*, in the course of generations into another type, *Artemia milhausenii*, by lessening the salinity of the water; and conversely, by increasing it. The results are open to criticism on several grounds, but it is enough to recognise that Schmankewitsch experimented with a *progressively changing environment* on a series of generations, and that the result is interpretable as due to modifications hammered on each successive generation without there being any inheritance of these modifications. It is also possible that the reproductive cells before or after liberation

were directly affected by the continuous change of salinity. (5) The fact that negroes and Mongolians are relatively immune to yellow fever has been cited as proof positive of the inheritance of an acquired character. But it may be that the quality of immunity was originally a congenital peculiarity, which has become dominant in the race by the elimination of those who were not immune. If it be objected that there are cases where a mother rabbit or guinea-pig has been artificially rendered immune to certain diseases, and has had young ones born immune, it may be answered that this is probably due to a kind of infection before birth, some antitoxin having passed from the mother to the unborn young. (6) The case of supposed modification-inheritance which has attracted most attention is that of Brown-Séquard's guinea-pigs. In a series of experiments extending over many years (1869-1891), Brown-Séquard showed that a partial section of the spinal cord, or a section of the sciatic nerve, was followed after some weeks by a peculiar morbid state resembling epilepsy. The offspring of the animals operated on were frequently decrepit, and a certain number showed a tendency to the so-called epilepsy.

As the original state may also be induced by bruising the sciatic nerve without cutting the skin, or by striking the animals on the head with a hammer, it seems unnecessary to consider the suggestion that the influence on the offspring was due to microbic infection. As a similar state may be induced in the dog by injury to the cerebral cortex, and was said in this case also to reappear in the offspring, it seems that we have not to deal with a peculiarity of the guinea-pig alone. As the epileptic state does not occur spontaneously in guinea-pigs, we may exclude the possibility of coincidence and the suggestion that captive guinea-pigs are nervously morbid. As the tendency to epileptic fits (which did not last long) was seen only in the offspring of animals which had been operated upon, and was only manifested after appropriate stimulus, especially after irritating an "epileptogenic" zone behind the ear on the same side as the original injury, we may exclude the suggestion that the epileptic tendency was imitative. Brown-Séquard's results have been confirmed by Dupuy (1890), Westphal (1871), Obersteiner (1875), and Romanes (1895); and no contrary evidence has as yet set aside the suggestion of modification-inheritance which the case conveys.

On the other hand, it should be noted that the results are very various. In one set of experiments (Obersteiner, 1875), out of thirty-two young ones born of artificially epileptic parents, only two showed symptoms of epilepsy. The results in Brown-Séquard's cases were very various,—general feebleness, motor paralysis of the limbs, trophic paralysis resulting in loss of toes, cornea, etc., and only in some cases epilepsy or some similar nervous disorder. It seems fair to say that what was inherited was decrepitude; the modification was too violent to be a fair case; it disturbed the whole organism, nutritive and reproductive functions alike, and was thus almost bound to cause abnormality in the offspring.

Although the evidence suggesting the transmissibility of acquired characters seems altogether inconclusive, it may be noted that somatic modifications have enormous individual importance. If they are not transmissible, the importance of securing good "nurture"—both functional and environmental—is rather increased than diminished. It has also been pointed out by several naturalists, that although modifications may not be of direct value in evolution (if they are not transmissible), they may be of great indirect value by acting as the fostering nurses or shields of congenital variations in the same direction. Thus, if we suppose swarthiness to become a condition of survival in a given country, there would probably be some inhabitants with a strong natural or congenital tendency in this direction; there would probably be others in whom the congenital variation towards swarthiness was weak and incipient; there would probably be others who had simply a susceptibility to acquired swarthiness; and there would probably be others who were persistently blonde. The first would in the course of natural selection tend to become in themselves and in their progeny the dominant stock; the last would tend to be rapidly eliminated; but it is conceivable that those who made up for their lack of natural swarthiness by a great susceptibility to acquired swarthiness would also be very successful; in other words, it is conceivable that the modification, though never taking heritable root, would serve as a life-saving screen until coincident congenital variations in the direction of swarthiness had time to grow strong. The case is hypothetical, but the idea is applicable to realities.

Telegony. — The term telegony has been applied to the doubtful, certainly rare, but if true, very remarkable occurrence of cases where an offspring resembles not so much its father as a previous mate of its mother. In other words, telegony is the supposed influence that a male may, through effective impregnation of a female, exert on offspring subsequently borne by the same female to a different sire. To take a simple instance, the race-horse "Blair-Athol" had a very characteristic blaze or white bald face; and it is said that mares which had borne foals to "Blair-Athol" subsequently produced to quite different stallions foals with the "Blair-Athol" blaze. The alleged cases are of much interest, but many of them are ill-authenticated, and others are capable of being differently interpreted. At the same time some explanation

must be found for the fact that a belief in telegony is widespread among practical breeders.

Telegony has been alleged to occur in man, horses, cattle, sheep, pigs, dogs, etc., and even in pigeons, but even the most circumstantially recorded cases, such as Lord Morton's Arab mare, are far from satisfactory, and the majority cannot be called scientific data at all. In his "Penycuik Experiments" Cossar Ewart (1899) proved this at least, that telegony does not generally occur even when favourable conditions are afforded; only in a very small percentage of cases was there anything even suggestive of telegony. Moreover, where peculiar phenomena of inheritance were observed they seemed to be readily explicable on the "reversion hypothesis." The general nature of the experiments may be illustrated by one of the best cases. A Rum pony mare "Mulatto," of remarkably pure breed, was served by a Burchell zebra stallion "Matopa," and the result (in August 1896) was "Romulus," whose markings were quite different from those of his sire, being suggestive rather of the Somaliland zebra. In 1897 "Mulatto" had a bay colt foal to a grey Arab stallion, and this foal—unfortunately short-lived—gave no evidence of telegony. The stripes which most frequently occur in horses were absent; there were others which are not uncommon in horses; but the most distinct markings (not that any were strongly developed), namely, those across the croup, were of a sort extremely rare both in foals and horses. In short, the marking of "Mulatto's" second foal was puzzling, but in no definite way suggestive of the influence of the previous zebra sire. In this, as in other cases, the verdict as to the occurrence of telegony was non-proven. It is evident, however, that *if* telegony be a reality which occurs very rarely and under peculiar conditions, the probabilities are many against an experimenter realising these conditions in a short time; and therefore it is interesting to notice that Karl Pearson, working by statistical methods, was unable to find any quantitative evidence of a steady telegonic influence in man.

Till we are sure of the facts to be explained, the discussion of interpretations is gratuitous, but a brief summary may be given. (*a*) It has been suggested that the phenomena are simply illustrations of reversion, and that the resemblance between the offspring and a previous sire with which it has no genetic relation is a coincidence. The plausibility of this explanation will vary in different cases. Thus, Finn points out that the occurrence of feather-legged fowls from pure Dorking parents, or of polled lambs from black-faced horned parents, cannot be set down to reversion, "feather-legged fowls and polled sheep not being ancestral types." All depends, however, on the proved purity of the parent breed. (*b*) Another theory interprets telegony as due to maternal impression, the supposition being that the mental image, etc., produced in the mother by the first sire exerts an influence on subsequent germs, or on their development by another sire. But *see* "Maternal Impressions." (*c*) Weismann and others have suggested that spermatozoa of the first sire may reach the ovary, and become associated with immature ova, or may be in some way stored; and that a belated fertilisation may coincide in time with a second coitus by a different sire, to which the offspring would be naturally referred. Were this the case, we should expect to find cases where offspring were produced without any second sire at all, and no such cases (among higher animals at least) are known. (*d*) Somewhat subtler is the suggestion—often called the "infection hypothesis"—that the seminal matter of the first sire may, apart from the fertilisation of an ovum or ova, influence the reproductive organs or the constitution of the mother in such a way that subsequent gestations (following impregnation by another sire) may be affected. The probability of some physiological influence is probable, but it is difficult to conceive that the influence should be of so precise a nature as to evoke in offspring by a second sire a resemblance of the first. (*e*) Perhaps the most plausible theory is, that the mother is influenced through the fœtus during pregnancy, and that the influence reacts on subsequent offspring. This so-called "saturation hypothesis" suggests that some of the characteristics of the sire, while expressing themselves in the development of the embryo, may, as it were, saturate into the dam, and affect her constitution in such a precise way that her offspring by subsequent sires may through maternal influence be affected with some of the characteristics of the first. Thus, Sir William Turner (1889), in discussing Lord Morton's case, says: "I believe that the mother had acquired during her long gestation with the hybrid the power of transmitting quagga-like characters from it, owing to the interchange of material which had taken place between them in connection with the nutrition of the young one. . . . In this way the germ-plasm of the mother, belonging to ova which had not yet matured, had become modified whilst still lodged in the ovary. This acquired modification had influenced her future offspring derived from that germ-plasm, so that they in turn, though in more diluted form, exhibited zebra-like markings." It is conceivable that something like this may occur in the case of a poison or protective antitoxin, which might diffuse in and out. We can imagine that a sire infected with some virulent disease, and showing certain structural disturbances associated therewith, may have offspring which are similarly affected, and that the influence from them may pass before their birth into the constitution of the mother, and so affect her that subsequent offspring by a healthy sire are diseased after the

manner of the first. But while we have some facts to go upon in regard to the diffusion of toxins and antitoxins, we have none as yet which warrant us in supposing the diffusion of structural characteristics or of the germinal representatives of these.

It remains to ask for some explanation of the widespread belief in the occurrence of a phenomenon, the scientific evidence for which seems so slender. There is no doubt, we are told, that the value of a pure-bred bitch at once goes down if she has been accidentally lined by a mongrel, and it is possible that there may be good reason for this, apart from the fact that the episode is not one which figures well in the books. It is possible that the constitution and temper of the bitch may be subtly affected by a coitus—especially fertile coitus—with a dog of inferior strain, and that the deteriorated constitution may react upon future offspring although real telegony does not ensue. It is hardly sufficient to remind ourselves that people are indescribably careless about their scientific beliefs, and that breeders are often too superstitious, for considerations of money value have a potent effect in evolving carefulness, and breeding is gradually becoming an art based on scientific conclusions. There must be some basis for the widespread belief, and the answer given by the practical men themselves is that they have abundant experience of telegony. This leads us to look for phenomena which might be readily mistaken as telegonic, and there can be little doubt that Ewart is right in thinking that the mistake is in the misinterpretation of *reversions*. The unexpectedness of results when different races are crossed is well known. A dark bantam hen, paired with an Indian game Dorking, produced, amongst others, a cockerel almost identical with a jungle fowl (*Gallus bankiva*), that is, with the original wild stock. What occurs when different races or breeds are crossed may occur on a smaller scale when individuals of the same breed, but of different strains, are crossed. Reversionary phenomena, however they may be theoretically interpreted, are frequent, and when they occur they are to the practical breeder usually disappointing. In search for an interpretation, he sometimes thinks that he finds one in telegony; that is to say, he gives the blame of the reversionary phenomenon not to the immediately preceding crossing, which may have been theoretically correct, and should have turned out well, but to some remoter, less careful, or perhaps accidental crossing. In this way the remoter sire is made the scape-goat for the reversion, and the belief in telegony has grown.

Inheritance of Disease.—The study of the heritability of diseased states has not disclosed any general fact which is not observable in normal cases; and it is only for convenience that the two aspects should be separated. Since Lucas (1847) collected his data showing the heritability of malformations, numerical abnormalities, and many diseased states of body and mind, a more critical study has led physicians to formulate a number of distinctions between real and apparent inheritance. As these make for progress they may be briefly illustrated. For notes on the heritability of particular diseases the separate articles should be consulted.

(*a*) The reappearance of a diseased condition in successive generations does not prove that it has been transmitted, or that it is even transmissible. The Alpine plants which Nägeli brought to the botanical garden at Munich became so much changed that they were hardly recognisable as the same species, and their descendants were likewise transformed. There was no doubt as to the reappearance of the unusual characters, but there was every doubt that the reappearance was due to inheritance. That it was due to the persistence of the new conditions, and to the changes which these directly impressed on each successive crop, was shown by the fact that when the plants were removed to poor, gravelly soil, the southern modifications disappeared, and the plants were retransformed into their original Alpine state. So it is with many diseased states which reappear generation after generation, not because they have been transmitted, but because of the persistence of the unhealthy stimuli in function or in environment which originally evoked them.

(*b*) Even when a child is born with symptoms or definite expressions of a disease, it does not follow that the disease was part of the inheritance. It may have been acquired by infection through the mother during the fœtal period. This may be illustrated in some cases of syphilis. Similarly, there is a growing body of evidence to show that in certain mammals (if not also in man) there may be a passage of antitoxin substances from the blood of an artificially immune mother to the blood of the fœtus, so that the offspring may be in consequence born immune. But no one who thinks clearly would call this a case of inheritance.

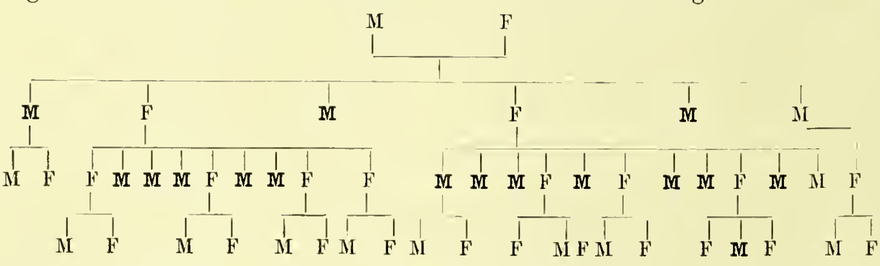
(*c*) In many cases it seems possible and useful to draw a distinction between the inheritance of a definite disease and the inheritance of a constitutional predisposition to it. Thus, since tuberculosis is a bacterial disease, since few children are born tuberculous, and since the disease attacks unequally those who are equally exposed to the same external conditions of infection, it seems probable that what is inherited is a blastogenic variation which expresses itself in "vulnerability of the protective epithelia," etc., in short, in a deteriorated power of resistance to the tubercule bacillus. In the same way, to take a case apart from bacterial infection, it seems probable that gout is not as such transmissible, but that

what is inherited is a blastogenic variation which expresses itself in an altered mode of eliminating nitrogenous waste,—a constitutional vice which becomes more apparent through excess of food and alcohol.

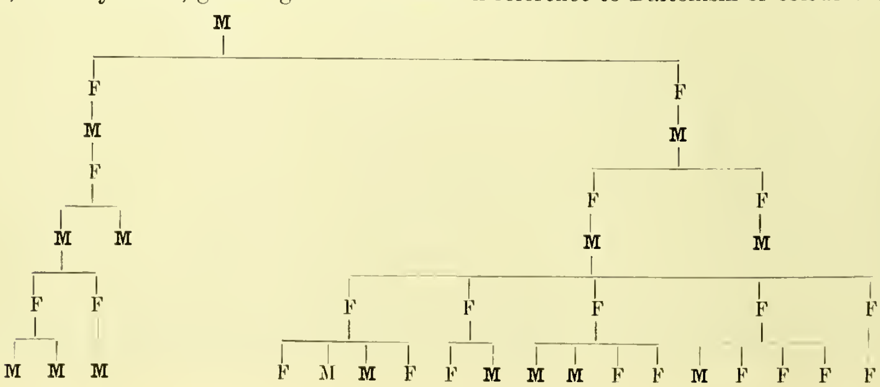
That diseased states of the nervous system run in families is undeniable, but in many cases they change in particular expression from generation to generation. This points to the position which many hold, which is well argued for by Rohde (1895), that what is really inherited is a germinal variation, which may express itself in general neurasthenia, easy exhaustibility, etc., or under sufficient provocation in some form of acute neurosis. There is no clear case of a normal subject becoming an acute maniac through external shock and transmitting his disease; and Rohde's conclusion, after a careful survey, is that all transmissible nervous diseases have a germinal origin. As Clouston has said: "A neurotic heredity is seen to resolve itself into general morbid tendencies rather than direct proclivities to special diseases." What we have said does not imply that persistent nerve-fatigue and neurasthenia in parents may not favour the outcrop of neurosis in the offspring, for the abnormal nervous condition may through nutritive disturbances affect the germ-plasm (as even Weismann admits), and the fœtus may be readily affected disadvantageously through the mother.

(*d*) The same, or a closely similar diseased state, may arise in different ways, and the heritability will differ with the mode of origin. If the diseased state is inborn in the strict sense, if it be the result of a blastogenic or germinal variation, the probability of transmission is great. But if it has been induced adventitiously by external influences the probability is slight. The distinction is a real one, but it is not always readily drawn in actual practice. Thus the difficulty of distinguishing congenital deafness from that which is adventitious—the result, for instance, of various infectious diseases—may, perhaps, explain why in E. A. Fay's statistics (3078 marriages, 6782 children), the percentage of deaf children in families where both parents were deaf was 8·458; where one parent only was deaf the percentage was larger, namely, 9·856. Where both parents were believed to be congenitally deaf the percentage of deaf children was 25·931; where one parent was deaf congenitally and the other adventitiously, it was 6·538; where both parents were adventitiously deaf, it was only 2·326. Where one parent is congenitally deaf and the other hearing, 11·932 per cent of the children were deaf; where one parent was adventitiously deaf and the other hearing, the percentage was 2·244. In short, it is not evident that adventitious deafness is inherited. Fay's statistics show that deafness among the relatives of the parents increases very largely the likelihood of there being deaf children; and they also seem to show that consanguineous marriages greatly increase the probability of the inheritance of deafness, or of the tendency to it.

(*e*) In many cases there is abundant evidence of the transmission of a specific disease, but this seems always to be what the biologist would call a disease of germinal or blastogenic origin. Thus we may cite a case of hæmorrhagic tendency given by Klebs (quoted by Turner, 1889), where the dark letters stand for affected subjects (all males); it illustrates first a diffusion and then a waning of the disease.



Horner, cited by Klebs, gives a good illustration in reference to Daltonism or colour-blindness.



What has just been noted in regard to specific diseased conditions is likewise true of many abnormalities. Polydactylism has been traced through six generations, and Turner notes a case where a shortening or imperfect growth of the metacarpal bone of the ring finger of the left hand was "traceable throughout six generations, and perhaps even in a seventh, and was, as a rule, transmitted alternately from the males to the females of the family." Scores of examples might be given.

To sum up this section, we may cite four general conclusions from a recent discussion of the subject by D. J. Hamilton:—

(1) There is no evidence proving that diseased conditions of body, excited by external agencies, using the term in its broadest sense, can be transmitted hereditarily through generations.

(2) The various hereditary tendencies or predispositions to disease of the hereditary type have arisen as variations in the germ-plasm.

(3) These predispositions to disease probably extend far back into the history of the human race, and break out only occasionally in accordance with the laws of atavism. [Others would say that the great abundance of presently occurring germinal variations renders it unnecessary to seek for an origin in the distant past.]

(4) External agencies are merely the means of bringing them to light.

When all is said there remains no doubt that morbid conditions and predispositions are inherited, and against this we have to place the probability that relative immunity is also becoming heritable. In the course of natural selection, keenest during the early years of life, the less immune tend to be eliminated, and the standard is thus raised. But in this struggle, as Reibmayr has argued, the most momentous factor is in the external conditions of function and environment, for if these favour the morbid inheritance the organism has to fight a battle with two fronts, which is seldom hopeful. The hope is in the slow increase of constitutional immunity on the one hand and in the securing of wholesome conditions of life on the other. But another side of the problem must not be overlooked, which Haycraft has emphasised in his *Darwinism and Race Progress*—If the race eliminates its own eliminators (the disease germs) which have at least helped to make it what it is, or if it becomes no longer susceptible to their eliminative action, what selective agents —even more discriminating, we may hope—are to take their place?

An article on this subject at the present date ought to close with a reminder that in regard to many problems of heredity and inheritance we are far from being able to formulate general conclusions, and that the hopeful outlook is not in theorising, but in experiment, in the collection of precisely observed data, and in the skilful use of statistical methods.

Hering's Theory.

See Colour Vision (*Theories of Colour Perception*).

Hermaphroditism.

See also Generation, Female Organs of (*Arrested Developments*); Ovaries, Diseases of (*Hernia*); Scrotum and Testicle, Diseases of (*Abnormalities, Cleft Scrotum*); Uterus, Malformations of.

Hermaphroditism, in the strict and ancient sense of the term—the existence, namely, in the same human being of functionally active testicles and ovaries—has not yet been met with; the name, however, is applied to the cases in which glands anatomically ovaries and glands anatomically testicles have been found in one individual, or, much more commonly, to the cases in which the true sex is masked or rendered dubious by the existence of malformations. There are, therefore, two distinct groups of cases, the first of which contains the anatomically true hermaphrodites (hermaphroditismus verus), while the second includes the individuals of doubtful sex (hermaphroditismus spurius). Of these two groups the latter contains by far the majority of the reported cases which have come to the test of the post-mortem room table and the microscope.

True hermaphroditism may be of three kinds —lateral, bilateral, and unilateral. *Lateral* hermaphroditism may be defined as the presence of an ovary on one side of the body and a testicle on the other, and of this it is claimed that some certain cases have been met with. With regard to that reported by Cramer in 1857, it is stated that there existed a rudimentary uterus and vagina along with, on the right side, a normal ovary, parovarium, and tube; and on the left side a tube, parovarium, and a supposed testicle lying in the scrotal sac. As, however, the microscopical appearances of the last-named body are not given, it is as reasonable to regard it as a herniated ovary. Schmorl's case is better established: it was that of a hypospadiac individual, 22 years of age, in whom, after operation, a small swelling appeared in the left groin, which, when excised, was found to be an ovary; the patient died, and at the autopsy it was discovered that there was a uterus bicornis, and on the right side a testicle with a rudimentary epididymis in the scrotum. Of *bilateral* hermaphroditismus verus, which may be defined as the presence of both an ovary and a testicle on both sides of the body, it must be regarded as doubtful whether a well-established instance has yet been reported. In Heppner's case, a premature, and, in other ways, malformed infant,

the external organs were those of the female; while internally there were a rudimentary uterus, a rudimentary vagina, a normal ovary, parovarium, and tube on both sides, and near to each ovary a body containing tubules running towards the hilum, and supposed to be a testicle. *Unilateral* true hermaphroditism must be very rare; in it there are supposed to be a testicle and an ovary on one side of the pelvis, and a testicle or an ovary or neither on the opposite side. Blacker and Lawrence claim to have described the only well-authenticated and genuine case of hermaphroditismus verus unilateralis; but it is to be remembered that it was in a fœtus and not in an adult. The fœtus was well formed save for the genitals; there was a uterus unicornis, a normal tube and ovary on the right side, and on the left side an ovo-testis, with a vas deferens and epididymis. Of course, the whole importance of the case rests upon the demonstration of the so-called ovo-testis; it is claimed by the authors that the gland on the left side was double in nature, that one part of it was ovarian and the other testicular. The microscopical appearances show that in one part there were cell-columns and Graafian follicles with a large amount of stroma, while in the other were tubules filled with cells, forming at the hilum a rete-like structure. Manifestly it becomes very difficult to decide as to the nature of a gland like the testicle or ovary, which in early fœtal life is so similar in structure; arrested development of the testicle might leave a gland not unlike a poorly developed ovary.

Pseudo-hermaphroditism, or hermaphroditismus spurius, is much less rare than the true form; and the explanation is to be found in the embryology of the genitals. Pseudo-hermaphrodites are usually individuals in whom a part of the organogenic scaffolding of the genital organs, which is common to both sexes, persists instead of atrophying; thus in *male* pseudo-hermaphrodites there may be found coexisting the testicles and a uterus and tubes, the Müllerian ducts which ought normally to have atrophied having persisted. But there is never a stage in the embryo in which the scaffolding of both ovaries and testicles exists; hence the rarity, perhaps the impossibility, of true hermaphroditism. The other form of spurious hermaphroditism, pseudo-hermaphroditismus *femininus*, includes the cases in which an individual with ovaries (a woman therefore in reality) has, through adhesion of the labia pudendi, hypertrophy of the clitoris, and a hirsute development on the face, taken on the external appearances of the male. This form, or gynandry, as it is sometimes called, is not so common, and does not carry with it the same social dangers as pseudo-hermaphroditismus masculinus or androgyny; it requires no further description.

Male pseudo-hermaphroditism may be of three kinds. It may be *internal*, when there are testicles in association with external genitals of the male type, and a uterus, vagina, and even tubes. It may be *external*, when there are testicles along with female external genitals and a feminine build of body. Finally, it may be *complete*, when, in addition to testicles, there is a uterus with tubes and external genitals resembling the female type. The commonest form met with is that in which there is scrotal hypospadias (the urethra opening at the base of an imperforate and stunted penis), non-descent or incomplete descent of the testicles, and want of union of the two halves of the scrotum in the middle line. The resemblance to the female type may be intensified by the presence of a vulvar or rather a vestibular canal of no great depth, but simulating the vaginal orifice, and sometimes possessing a hymeneal membrane. Such individuals are usually registered at birth as females, and it is only when puberty arrives that the non-appearance of the menses, and the development of hair on the face and chest, along with other secondary male characters, throw doubt on the real sex of the person, and may lead to a medical examination. In the absence of such an examination it may unfortunately happen that the hypospadiac man is married as a woman, and Neugebauer has reported a number of cases of this occurrence. As in all other malformations, so in this, family prevalence (the occurrence of more than one case in the same family) may be met with, and both Croom and Chiarleoni have given details of supposed sisters who turned out to be hypospadiac brothers. In order to avoid the awkward consequences which may and do follow such erroneous declarations of sex, Lawson Tait has advised that when any doubt as to sex exists at the time of birth the child should be brought up as a boy; if this be done, the risks of error and the dangers resulting from it are much diminished, for male pseudo-hermaphrodites are more common than female, and individuals reared as males are less likely to enter the married state in ignorance of their true sex. Castration has been suggested and carried out in some instances, but it is of doubtful justifiability; redeclaration of sex is attended with many difficulties, as was abundantly shown in Croom's cases (*Trans. Edin. Obst. Soc.* xxiv. 102, 1899); but it is surely preferable in most cases to surgical procedures.

Hermite Process.—A means of disposal of sewage by subsidence. Nascent oxygen is obtained by passing an electric current through sea-water, and the fluid applied directly to the drains or sewers.

Hernia.

General.

See also APPENDIX VERMIFORMIS (*Hernia*); BLADDER, INJURIES AND DISEASES OF (*Malpositions*); BRAIN, SURGERY OF (*Hernia Cerebri*); CHEST, INJURIES OF (*Diaphragmatic Hernia*); CRETINISM (*Description, Umbilical Hernia*); DIAPHRAGM, SURGICAL AFFECTIONS OF (*Hernia*); FALLOPIAN TUBES (*Hernia of*); GASTRO-INTESTINAL DISORDERS OF INFANCY (*Constipation, Causes*); INTESTINES, SURGICAL AFFECTIONS OF (*Intestinal Obstruction, Internal Herniæ*); MUSCLES, DISEASES OF (*Hernia*); PELVIS, PERINEUM AND PELVIC FLOOR (*Rectocele, Prolapsus Uteri*); PERITONEUM, ACUTE PERITONITIS (*Etiology*); PREGNANCY, AFFECTIONS OF GENERATIVE ORGANS (*Hernia of Gravid Uterus*); SCROTUM AND TESTICLE, DISEASES OF (*Malposition of Testicle*); SCROTUM AND TESTICLE, DISEASES OF (*Impotence, Causes*); UMBILICUS, DISEASES OF (*Hernia*).

A HERNIA (ἔρνος = a branch) may be defined as the protrusion of any viscus through an abnormal opening in the walls of the cavity containing it. In general use the word "hernia" is tacitly understood as referring to an abdominal hernia.

In an abdominal hernia the viscus generally derives a complete covering from the peritoneum, and remains always covered by the skin. There are certain regions which from their anatomical conformation, from congenital incompleteness of development, or from both, are especially predisposed to the occurrence of hernia. These are the inguinal, femoral, and umbilical. Hernial protrusions may also occur in the following situations:—at the obturator foramen, in the perinæum, at the sciatic notch; in the lumbar region and at the linea alba or any portion of the anterior abdominal wall. All such herniæ are known as *external*, in contrast to the *internal*, which occur within the peritoneal cavity, generally into certain pre-existing peritoneal fossæ.

A hernia consists of (*a*) the sac, (*b*) the contents, (*c*) the coverings.

(*a*) *The Sac* consists of peritoneum continuous with and derived from the parietal peritoneum. As a general rule the sac is complete, but in certain cases, for example, "extraperitoneal" cystocele, it may be absent, and in others, such as "paraperitoneal" cystocele, it may be incomplete. Two forms of sac are recognised:—

I. The Congenital, and II. The Acquired.

I. *The Congenital Sac* results from the want of closure of peritoneal processes normal in the fœtus. Such are the *processus vaginalis*, the canal of Nuck, and the peritoneal protrusion at the umbilicus. These will be subsequently referred to in detail. Some forms of sac produced by the traction of an aberrant attachment of the gubernaculum must also be considered as congenital. Such is the sac of an infantile hernia, and such, probably, is the sac formed from one of the pouches of Rokitansky.

II. *The Acquired Sac*, in its earliest stage, is represented by a mere laxity or bulging of the parietal peritoneum. Then at a certain point, the internal abdominal ring, for example, a dimple is formed which, gradually deepening, forms a tube-like process lying in the inguinal canal. The orifice of communication with the peritoneal cavity is now the widest portion of the sac. As the tube-like structure gradually elongates it reaches the upper part of the scrotum, and there, relieved of pressure, expands readily and rapidly, and becomes spherical in shape. To some extent the increase in size of the sac may be the result of the dragging down of peritoneum from the iliac fossa, but it is largely due to the inherent capacity for expansion under a process of stretching possessed by the serous membrane. The upper end lying within the canal is then the narrowest portion of the sac, and is known as the *neck*, the expanded lower end being known as the *fundus*. During the time of the progressive enlargement of the sac adhesions form between it and the structures immediately external, so that when the sac has reached even a moderate size it is fixed, and, except under unusual circumstances, irreducible. The neck of the sac is generally described as being thrown into folds which, from irritative inflammation, adhere to one another, and result in the formation of a constriction, a thick fibrous band, at the abdominal orifice. Such a condition is certainly rare. Since the introduction of the operations of Bassini and Halsted, which impose upon the surgeon the necessity of opening up the inguinal canal and laying bare the neck of the sac, it has been noticed that even if any pleats are present at the abdominal orifice they unfold themselves as soon as the external pressure is relieved. In not a few cases, however, the sub-peritoneal tissue is thicker at the neck than elsewhere, and by a local condensation may form a fibrous

ring, but such thickening does not usually affect the peritoneum.

In most cases the interior of the sac is quite smooth, but occasionally adhesions may form, as the result of inflammatory processes, and give rise to irreducibility of the hernia.

If the adhesions affect the mouth of an empty hernial sac and result in its closure there, or if a plug of omentum adhere in the neck of the sac and effectually block the orifice, fluid may accumulate in the interior of the sac so cut off and a *hydrocele of the hernial sac* results.

The sac wall is, in most examples, of the same appearance and thickness as the peritoneum from which it is derived. In old-standing herniæ, especially if an ill-fitting truss or one exerting ill-regulated pressure has been worn, the sac will be found thickened, tough, fibrous, or almost cartilaginous in density.

(*b*) *The Contents.*—Any of the abdominal organs, including the pancreas, may be present in a hernial sac, but in many of the operations undertaken for the radical cure the sac is found empty. The most usual content is probably the ileum, then the omentum, and the jejunum. The large intestine, especially the sigmoid, is not infrequently seen. The omentum generally lies in front of the intestine if both are present. It exhibits a marked disposition to the formation of adhesions to other structures contained within the sac. Adhesions present in a hernial sac may run

1. Between one portion of the sac and another,
2. Between omentum and the sac,
3. Between omentum and intestine,
4. Between intestine and intestine,
5. Between intestine and the sac.

The last are decidedly rare. A thin tag of omentum adherent to the neck of the sac is frequently found on laying open the inguinal canal. Unless the portion of the sac in the inguinal canal be freely opened up, these little tags or threads of adherent omentum are apt to be overlooked. A persisting adhesion of such a kind is not improbably a potent factor in the recurrence of a hernia after operation.

The omentum undergoes certain changes when it has been for any length of time in a hernial sac. It becomes thickened, tough, fibrous and brawny, and in some instances considerably increased in bulk, forming then a hard globular mass connected with the intra-abdominal portion by a more or less narrowed pedicle. Cysts may form, or a calcareous deposit occur, or rarely, malignant disease or tubercle develop. A second spurious sac may be formed by a complete envelopment of the gut in the sac by a thin and stretched omentum. Holes or gaps in this structure have allowed bowel to pass through them, and strangulation has thereby resulted. If intestine alone is present the hernia is known as an *enterocele*, if omentum alone as an *epiplocele*, if both as an *entero-epiplocele.* If only a portion of the wall of the intestine is included in the sac the condition is known as *partial enterocele*, or Richter's Hernia. This is most frequent in femoral hernia. A hernia of Meckel's diverticulum is known as *Littre's Hernia* (not Littré, as frequently written). Other adventitious bodies are occasionally found in hernial sacs, the commonest of these being formed from detached appendices epiploicæ, or tags of omentum which become coated with a thin tunic of fibrous tissue.

(*c*) *The coverings* of a hernia naturally vary with the region in which the rupture exists, but in the same region the conditions presented may vary considerably. Thus in the inguinal region a small hernia, especially a recent one, which has not suffered the pressure of a truss, will reveal on examination all the layers recognised by the anatomist. In an older, larger hernia, where the skin is thickened or chafed by the wearing of a truss, the layers are indurated, fused, and not clearly separable. In the umbilical region, owing perhaps to unrestrained bulging on the part of the contents, the skin becomes thinned, shiny, and almost translucent. In not a few cases ulceration from pressure or friction may extend widely and lay bare the sac and its contents.

Causation of Hernia

The causes of hernia are many. There is no single determining factor. The difficulty is not to explain the occurrence of hernia, but rather to understand how any human being goes through a reasonable length of life without suffering from rupture. It is almost impossible to find any subject in the post-mortem room of which it is justifiable to say that in him or her a condition of hernia might not very readily have existed. In all bodies one or more of the "causes" are present. For convenience of description the various conditions leading to hernia may be considered as *predisposing* and *exciting*, or *determining*.

Predisposing Causes.—1. Beyond question the most frequent of these is a congenital aberration of development. Such aberration may affect:—

(*a*) *The Vaginal Process.*—This offshoot from the peritoneal cavity may remain patent or may undergo only partial closure. The "physiological fusion" leading to the obliteration of the process begins earlier and proceeds more rapidly on the left side. Congenital hernia and similar forms of hernia are therefore more commonly noticed on the right side. Probably the partial or complete want of closure of the process and the consequent formation of hernia is far more frequent than is generally conceded.

(*b*) *The Descent of the Testis.*—Any of the forms of retention or ectopy of the testicle strongly predispose to hernia.

(*c*) *The Gubernaculum.* — Irregular attachments of the gubernaculum may pull down into the scrotum accessory sacs, as in infantile hernia. The pouches of Rokitansky seen in the peritoneum, near the internal abdominal ring, may also be formed in this manner. The descent of a congenital cæcocele is induced by traction of the gubernaculum.

(*d*) *The Abdominal Wall.*—Congenital gaps may exist at the umbilicus or at any portion of the linea alba. Congenital weakness of the lower part of the abdominal wall, associated more especially with retention of the testis, is recognised by Macready.

2. *Heredity.* — The children of herniated parents are more likely than others to suffer from hernia. Macready, who has especially investigated this point, concludes that "inheritance is an agent, though perhaps a remote agent, in the production of hernia, and that the influence of the two sexes is nearly equal."

3. *Sex.*—All writers are agreed that males are affected more frequently than females. According to Macready the following are the percentages:—Male inguinal, 83·5; female inguinal, 8·5; female femoral, 5·9; male femoral, 2·1.

4. *Age.*—Hernia may be met with at any age. In childhood and early youth congenital causes are at work. During the first year of life an extraordinarily large number of ruptures are seen. Then, year by year, fewer until puberty. From puberty till the termination of the most active period of physical life, at or about fifty years of age, the number steadily increases, but after the latter period gradually declines.

5. *Weakness of the Abdominal Wall.*—In addition to the congenital laxity already referred to, there may also be an acquired feebleness of the muscles. In many cases of inguinal hernia, especially in adults and old people, lateral bulgings of the abdominal wall between the outer edge of the rectus and Poupart's ligament are seen when the patient is standing. There may be also a central bulging of the two recti, and the appearance of *ventre à triple saillée* of Malgaigne is presented. Any form of injury to the anterior abdominal wall may result in the weakening of the damaged area and the protrusion of a viscus through it. A stab, an incision made during an operation, or a blow leading to rupture and subsequent atrophy of the muscle ("Guthrie's Hernia") may all originate a hernial swelling.

The rapid wasting of a large deposit of fat, the wasting associated with old age or with acute illness, may leave the abdominal wall incapable of resisting pressure from within. Parturition, ovarian tumours, ascitic effusion may, by the distension they cause, result in a weakening and a laxity of the whole parieties.

The importance to be attached to the hypogastric bulgings has, I believe, been considerably overestimated by Mr. Lockwood. He remarks: "This bulging always accompanies acquired hernia, and, I believe, precedes its occurrence." Because of this general laxity, Mr. Lockwood further teaches that "curative operations upon acquired hernia are to be avoided." It seems to me that this is an exaggeration both of the frequency of the condition and of the severity of it in relation to operative treatment. Mr. Treves remarks: "In any but aged or broken-down subjects this enfeebled condition of the belly-wall can be greatly improved or even overcome by suitable exercise of the abdominal muscles," a statement which I have verified on several occasions.

6. *The Condition of the Mesentery.*—The normal mesentery is of sufficient length to permit of the entrance of the bowel into an inguinal or a femoral sac. An elongation of the mesentery is consecutive to hernia, and not, as was generally supposed, the initial and responsible defect. Lockwood has shown that "in undoubted cases of acquired hernia, the length of the mesentery is the same as in unruptured people of the same age." As age progresses, the normal attachment of the mesentery to the posterior abdominal wall becomes altered by the gliding downwards of this structure. In its descent the other abdominal viscera accompany the mesentery in what Lockwood terms a compound "prolapse of the mesentery." A profile view of the abdomen of a patient affected with this condition shows a flattening or an excavation above, and a bulging below, the umbilicus. Such a state naturally predisposes very strongly to the onset of a rupture, and if, at the same time, the abdominal wall is lax and enfeebled a hernia is certain to occur. The two conditions are not infrequently associated, and Lockwood believes that both are the expressions of a general tissue deterioration. A rupture, he writes, is "in many cases the local manifestation of a widespread tissue-change."

Exciting or Determining Causes.—The immediate cause of a hernia is any condition which increases the intra-abdominal pressure.

1. *Effort* is the most frequent of these. In the lifting of heavy weights, in straining on coughing or on defæcation, or on micturition when stricture or prostatic enlargement exists, a strain is put upon the abdominal muscles and the cavity enclosed by them is lessened. In children, a tight prepuce, a stone in the bladder, intestinal irritation from bad feeding or from worms and so forth, produce persistent straining. Certain occupations, involving heavy work in certain postures, thus determine the occurrence of hernia. In general it may be said that those trades demanding the most severe exertion show the highest proportion of ruptured persons.

2. Intra-abdominal pressure may be increased by the growth within the cavity of any form of tumour, or by the accumulation of fluid in large

quantities. The bowel may, in this manner, be forcibly and powerfully expressed into a hernial sac, even when the patient is bedridden.

The Conditions of a Hernia

A hernia may be reducible, irreducible, incarcerated, inflamed, or strangulated.

Reducible Hernia.—*Symptoms.*—A patient, the subject of a reducible hernia of slow formation, will complain (often before any evidence of the hernia is present) of a sense of weight, uneasiness, aching or discomfort in the affected region. This is especially noticeable after exertion, and is relieved by rest. There is, however, a marked difference in individuals in this regard. It is no uncommon thing to see a workman going about his daily task unconcerned by the presence of a gradually enlarging rupture. On the other hand, a bubonocele, trivial and quite inconspicuous, will give rise to complaints of serious disablement. After a time there may be evidence of intestinal disorder, colicky pains, generally referred to the umbilicus or thereabouts, and dyspeptic trouble. Constipation, especially if the colon is involved in the hernia, may be a prominent, and is occasionally the chief, symptom. Loss of appetite, nausea, and general intestinal discomfort may from time to time be observed, and are the more readily produced by errors of diet, which, under normal conditions, would not be visited with punishment.

Signs.—A reducible hernia, wherever occurring, gives rise to a soft rounded swelling, generally smooth on the surface and regular in outline. On the patient assuming the erect posture, or on straining or coughing, the hernia descends, or, if already down, undergoes such an increase in size as gives rise to an expansile impulse. This "*impulse on coughing*" is present in all hernias, except the strangulated. It is most distinct in enterocele, less so in epiplocele, especially if irreducible, and least so in obstructed hernia.

When intestine is present, the tumour is quite soft and elastic and tympanitic on percussion if of moderate or large size. On applying an even pressure to the tumour it gradually lessens and eventually disappears within the abdomen, undergoing "*reduction.*" If gas and fluid are together present a bubbling sound will be elicited by the pressure and movement; on the reduction of the last inch or two of the bowel there is a feeling as though the gut were being drawn away from one's fingers; these two signs constitute the characteristic "slip and the gurgle" of an enterocele.

When omentum is within the sac, it lies, as a general rule, in front of the bowel. The hernia is then not so smooth, feeling perhaps knotted, lumpy, and irregular. Reduction is slower, and there is neither slip nor gurgle. When the omentum descends again it does so slowly, oozing out of the abdomen evenly and regularly.

Taxis.—For the reduction of a hernia a special manipulation, *taxis*, is employed. The patient is lying upon the back. If the hernia is inguinal or femoral the pelvis may be raised by one or two pillows and the thighs slightly flexed. The neck of the sac in an inguinal hernia, for example, is surrounded by the thumb and fingers of the left hand, and pressure is applied by them downwards towards the fundus to free the neck and to ensure that force applied by the right hand at the fundus does not lose itself in expanding the neck and the upper part of the sac.

Irreducible Hernia.—An irreducible hernia is one in which there is an impediment to the return of the bowel within the abdomen. Irreducibility entails a likelihood of other and more serious complications, inflammation, incarceration, and strangulation.

The causes of irreducibility are :—

(1) *Adhesions within the Sac.*—These in the majority of cases implicate the omentum, and one finds therefore that 90 per cent of irreducible herniæ are epiploceles. Adhesions may involve any of the contents.

(2) *Changes in the Hernial Contents.*—The omentum or mesentery may increase in bulk from a deposit of fat. Cysts in the omentum or mesentery; tumours of the bowel, simple or malignant; tubercular deposits in the sac or its contents, have all been observed as causes of irreducibility.

(3) *Large size of the Hernia.*—During the course of years a hernia may undergo steady increase, until there is more in bulk outside than inside the abdomen. The abdominal parietes naturally accommodate themselves to the altered conditions, and at the last cannot yield to a degree permitting replacement. The hernial contents suffer from "*perte de droit de domicile,*" as Petit very aptly expressed it. In 10,000 cases Berger found this the cause of irreducibility in 122. In men, inguinal herniæ are affected; in women, umbilical.

The *symptoms and signs* are those of a reducible hernia with the exception of the fixity of the whole or some portion of the hernial protrusion. Irreducibility by keeping open the inguinal canal invites and encourages the descent of further portions of the bowel or omentum. Impulse on coughing is present, though not so clearly as in a reducible hernia. Intestinal worry is often troublesome, and the transverse colon especially may suffer. Symptoms are decidedly more aggressive than in a hernia capable of easy reduction. Irreducibility is not necessarily a permanent attribute.

Treatment.—A hernia whose irreducibility is recent may by appropriate treatment be not infrequently returned within the abdomen.

Confinement to bed, restricted diet, careful regulation of the bowels, and the application of steady even pressure by an elastic bandage with occasional efforts at taxis, have met with fair success. If the patient will not submit to

stringent regulations, much good may be done in inguinal hernia by Kingdon's truss. This is in the form of a "hinged cup" and exerts a steady pressure on the herniated mass in a direction opposed to that of its descent. It must be worn night and day, and it possesses the "great advantage that it enables a man to continue at his work whilst his rupture is under process of treatment." In 128 cases recorded by Macready, reduction was effected in 88, sometimes in a few days, sometimes in two or three years. When reduction is accomplished the "hinged cup" is discarded, and an ordinary inguinal or a rat-tailed truss worn. In femoral hernia pressure may be kept up by a truss with a hollowed pad, but reduction in this class of case is neither so frequent nor so speedy as in inguinal hernia.

In cases where reduction cannot be effected some form of truss with an excavated pad, or with a "bag" made to fit the hernial swelling, may be worn, in the hope that a further descent of bowel may be thereby prevented. But whatever method be adopted it must be recognised that an irreducible hernia is a constant menace, and may at any moment assume a condition which jeopardises the sufferer's life. Unless, therefore, there are sufficient reasons to the contrary, such as kidney, chest or heart disease, ascites, and so forth, operative measures should be advised if only to render possible and comfortable the wearing of an ordinary truss. The operation for the radical cure of hernia finds its chief justification in cases of irreducible hernia.

Incarcerated or Obstructed Hernia.—Incarcerated hernia is one in which there is an impediment (*a*) to the return of the bowel within the abdomen; (*b*) to the passage of the intestinal contents along the bowel involved in the protrusion. There is in fact a *local constipation* in an irreducible hernia which becomes turgid and swollen, but retains an impulse on coughing. The condition is generally seen in the bulky herniæ of old people. The bowels refuse to act, there is loss of appetite, the tongue is furred and the breath foul. If unrelieved by enemata and the reasonable employment of taxis, operation must be resorted to or the symptoms of strangulation will slowly develop.

Inflamed Hernia—"Hernial Peritonitis."—In this condition there is a localised peritonitis affecting the hernial sac or its contents. The omentum is very generally the seat of this acute condition, which results in many cases from external injury, a blow, unrestrained efforts at taxis, or the pressure of a badly-fitting truss. The symptoms and signs are mainly those of local inflammation. The impulse on coughing can be elicited.

General malaise, vomiting, and constipation, if present, are of such slight severity that they can readily be differentiated from the similar but severer symptoms of strangulation. The onset of inflammation is gradual and is attended with fever of varying height. In strangulation the onset is sudden, the development of symptoms is rapid, and there is shock in place of fever.

The patient must be kept in bed, and enemata given until the bowels act well; hot fomentations applied to the hernia will give ease. The diet must for a time be restricted to fluids.

Strangulated Hernia.—A strangulated hernia is one in which there is an impediment (*a*) to the return of the bowel within the abdomen; (*b*) to the passage of the intestinal contents along the bowel involved; (*c*) to the circulation in the imprisoned contents. A hernia is made serious to life only by the possibility of strangulation.

In partial enterocele and in Littre's hernia the obstruction may not yet be complete, either so far as the mechanical conditions or the symptoms are concerned. In a strangulated epiplocele the second condition (*b*) is, of course, not present. Such a condition, however, is rare, and the case would probably be looked upon as an "inflamed" hernia.

Mechanism of Strangulation.—It cannot be said that the precise circumstances under which strangulation occurs in a hernia are properly understood. No question relative to hernia, not even the operative treatment, has had bestowed upon it such a wealth of words as this; and it is in surgery as in finance—much paper and much poverty may coexist.

An enquiry into the history of most cases (not, of course, cases of partial enterocele, to which the following explanation is not applicable) will reveal an appreciation by the patient of the sudden enlargement of the hernial swelling on exertion. The hernia "comes down bigger than it has ever done before." There is an immediate difficulty in reduction. The hernia is not only more bulky but also tighter, more tense, than it has usually been. In such an increased descent of gut there will also be a larger involvement of mesentery. The mesentery, especially if containing any, even a moderate deposit of fat, is more resistant, more solid, than the elastic and easily compressible gut. In the narrower portion of the neck of the sac (in the inguinal or femoral canals or elsewhere), which may be supposed to be adapted to the quantity of gut or omentum, or both usually traversing it, there will be only the same amount of room for a larger bulk of tissue. The softer and more readily compressible tissues will suffer, and these are the bowel and the veins of the mesentery. There will consequently be venous congestion, and, as Kader's experiments have shown, this will result in a gaseous distension of the bowel involved. Fluid poured out in greater or less quantity as the result of the vascular obstruction will increase the tension in the sac. In this way the condition of strangulation is brought about.

If this explanation were correct we should expect to find the point of strangulation at the

most resistant part of the upper end of the sac. In an inguinal hernia the most resistant part is the external abdominal ring; in femoral hernia the crural ring. It is at these two points, in my experience, that the obstruction most usually is found.

In reading the accounts of operations for strangulated hernia we are accustomed to meet with the statement that the obstruction is "at the neck of the sac."

As I have already remarked, a thick fibrous constriction at the neck, though occasionally existing in the old herniæ, is decidedly rare. In an inguinal hernia an operation carried out by any of the methods except Bassini's, Halsted's, or Lockwood's, would not permit an accurate examination of the uppermost part of the sac, and the term "neck of the sac" would probably be held to include anything in the inguinal canal. Now, if in strangulated inguinal hernia the aponeurosis of the external oblique be divided an inch or more above the external abdominal ring, and the fibres separated down to the ring, it can be demonstrated without the possibility of doubt that in the great majority of cases the strangulating factor is the ring itself. Immediately that is divided reduction becomes possible.

That the neck of the sac may sometimes produce strangulation seems clear from the description of specimens of reduction in mass, in which the whole sac has been reduced with the contents still strangled by the cordlike condition at the neck. Such a condition, however, is of extreme rarity. Berger, who investigated the question as to the point of strangulation very fully, so long ago as 1876, wrote that the neck of the sac was "rarely capable of forming a veritable stricture."

In femoral hernia the crural ring is the obstructing agent; division of Gimbernat's ligament generally permits of reduction. In umbilical hernia the obstruction is formed by the tight fibrous ring outside the neck of the sac.

In some, comparatively rare, examples, there may be an acute strangulation *within* the sac as the result of the passage of a loop of bowel through a hole in the omentum, as the result of a volvulus at the neck of the sac, or as the result of the nipping of the gut by an adhesion within the sac. Under these circumstances there is an acute obstruction in a hernial sac unconnected with the hernia as such.

Pathological Changes in the Hernial Contents as the Result of Strangulation.—When a hernia becomes strangulated, the venous channels become engorged. Blood can still enter by the thicker and less compressible arteries when it can no longer return through the veins. The congestion leads to an exudation of fluid, the colour and general quality of which depend upon the severity of the pathological changes in the bowel. The gut involved becomes blue and livid in appearance, and its colour deepens by degrees until it becomes rich purple, and finally black or ashen grey. Small extravasations appear as the result of the rupture of distended venules; in the mesentery the extravasated blood may form a solid slab of clot. The natural lustre of the bowel is retained for some time, but as the wall becomes thickened and œdematous, the serous membrane looks dull and turbid, and flaky masses of lymph adhere to its surface. Gangrene may affect the whole of the imprisoned loop, or just that ring of it subject to the keenest pressure at, or near, the neck. This point especially should receive careful examination after division of the stricture, and for that purpose the bowel above and below the snared loop must be pulled down. The indentation made by the stricture is then readily seen, and can if necessary be dealt with.

When the epiploon is involved, congestion may pass on to gangrene, but does so very rarely. When omentum is present in a hernial sac, adhesions almost always form, and by them blood-vessels may carry an alternative supply of blood.

The fluid in the sac is at first thin and serous, but becomes by degrees more and more deeply tinged with blood; turbidity is soon noticed, and as soon as the bowel wall is damaged there is an escape of the bacterium coli commune. In the fluid this organism flourishes, produces harmful toxic products, and gives rise to a peculiarly foul and penetrating odour. The character of the fluid is a good index to the amount of damage the intestinal wall has suffered.

In amount the fluid varies considerably. When bowel is in the sac, there is always a fair quantity, but when omentum alone is present, or when a solid viscus such as the ovary is present, the fluid is less. Macready asserts that in 33 per cent of cases of strangulation no fluid is present, an estimate which I consider to be greatly in excess of the truth.

As the fluid becomes more putrid it affects not only the sac, but the coverings of the hernia, which become red, inflamed, and œdematous, and may in rare cases go on to gangrene and the formation of a fæcal fistula, the clumsy and unpleasant result of "Nature's cure."

Bacteriological examinations of the fluid have been carried out by many observers. Clado has found the bacterium coli commune to be most frequently present, and in cases of death from strangulation has observed the organism in the spleen, liver, and kidneys, and in larger numbers and more frequently in the lungs. Barbacci has recognised Fränkel's diplococcus, and the staphylococci pyogenes albus et aureus and streptococci have also been found. Brentano asserts that after strangulation has existed more than twenty-four hours, micro-organisms are constant. Weichselbaum considers the diplococcus pneumoniæ as the occasional cause of acute general peritonitis.

Changes in the Bowel above and below the Hernia.—The bowel above the hernia becomes distended to a degree dependent in part upon the duration and severity of the obstruction. Certain changes occur in it, which have been carefully described by Kocher. The bowel becomes blue in colour from venous stasis, ecchymoses may form in the mucous membrane or beneath the serous coat, nutrition is altered and the protective influence of the epithelium is destroyed. Absorption of bacteria and of the toxic products of the intestinal contents begin from that moment, and the patient, unless relieved, may die either from general intoxication with symptoms of weakness of the heart and collapse, or from local intoxication with necrosis of the mucous membrane, beginning in the ecchymosed patches and going on speedily to ulceration and perforation.

The Symptoms of Strangulated Hernia.—The symptoms of strangulated hernia are very similar to those of acute intestinal obstruction. The first occurrence noticed is the forcible descent of a larger mass of hernia than is usual, or the decided increase of a rupture that is irreducible. This *accession of bulk* is attended with *pain* in the hernial swelling, chiefly at the upper part and in the abdomen near the umbilicus, at first and for a brief period intermittent, but soon becoming and remaining continuous. The pain is very acute, with exacerbations of a colicky nature; it causes the patient to be bent double, to sweat profusely, to become collapsed and faint, and it rapidly induces nausea and vomiting.

Vomiting is the chief and most important symptom. It is unvarying, distressing, and exhausting. At first the stomach is emptied, then the duodenum, the vomit being bile-stained; finally, the decomposing and fœtid contents of the distended gut above the obstruction are ejected. So pungent and putrid is this vomit at the last, that it is described as "fæculent" or "stercoraceous," implying that fæces are contained in the vomit. This is rarely, if ever, the case, the odour and appearance being in fact caused by the bacterial profusion in the over-distended bowel. Hiccough is sometimes present, and is an untoward symptom. The temperature as a rule is subnormal; the pulse is rapid, thin, and wiry. Constipation is absolute and persistent: enemata may empty the bowel below the obstruction, and a little "flatus" accidentally introduced by the syringe may be passed, but neither fluid nor solid leaves the hernial sac. Even in partial enterocele the same completeness of obstruction is observed in the majority of the cases, as the result it may be of reflex paralysis of the gut, or of kinking at the hernial orifice. In others there may be the passage of flatus and occasionally of the liquid contents of the gut. The abdomen is generally a little prominent, is very rigid and tender on pressure. No distended or contracting intestinal coils can be observed. The *appearance* of a patient suffering from strangulation is generally characteristic. There is at first an unhealthy almost livid flushing of the face, soon giving place to pallor with shrinking and "drawing" of the features, and a fixed and meaningless staring of the eyes. The face and limbs are covered with a sweat, and feel cold and clammy to the touch. There is a tinge of blueness in all the skin. The expression of the face becomes anxious, and speaks of terror and impending disaster. The body looks, and is, desiccated, shrivelled, parched for lack of any water. The tongue is dry, harsh, and brown: thirst is intolerable. The urine is diminished in quantity, and indican may be present. In cases of intense strangulation of the small intestine, albumin may also be found. According to Englisch it is present in two-thirds of the cases operated upon, and is a sign of some importance. The symptoms of toxic poisoning are seen in their most striking form, and in a state of gradually deepening collapse the patient dies.

The Signs of Strangulated Hernia.—The hernial swelling is larger, often considerably larger, than is usual. It is tense and hard, and irreducible. There is no impulse on coughing. Manipulation of the tumour is resented, owing to extreme tenderness. If strangulation persists, the surface becomes of a livid or dusky red colour, which gradually deepens until a black slough is formed. Emphysema may be felt, and perforation of the gut and of the skin may in the end lead to the formation of a fæcal fistula.

Treatment of Strangulated Hernia.—The hernial contents must be returned within the abdomen at the earliest possible moment. Reduction may be effected by means of taxis aided by the hot bath or an anæsthetic, or by operation. Taxis, unless employed with discrimination and for a brief period, is probably more harmful than operation.

False Reduction.—Taxis, if employed without constraint, or occasionally even when judiciously applied, is attended with some risks, such as injury, bruising, or laceration of the sac or its contents, or the forcing of infected fluid into the peritoneum. The most serious accident, however, is that known as *False Reduction.* I have elsewhere discussed this subject fully; it is not necessary here to do more than epitomise my conclusions. False reduction may be described as of the following varieties:—1. Reduction into a sac lying between the peritoneum and the transversalis fascia. This sac has probably received the bowel on many previous occasions. It exists in both the inguinal and femoral regions, forming the special class of hernia known as properitoneal or bilocular or the "displaced" hernia of Bryant. This sac may pass (*a*) inwards to the bladder, (*b*) backwards to the iliac fossa, or (*c*) outwards towards the

anterior superior spine. This form is probably by far the most frequent. In a very few cases, not five in all, the apparent reduction has occurred into one of the interstitial sacs beneath or superficial to the external oblique. Such a case was that under the care of Mr. Cock (Guy's Hospital Museum, No. 1120). 2. Reduction of the whole sac within the abdomen but outside the peritoneum, the relations of sac and contents to one another being unaltered and strangulation at the neck still persisting. It is probable that the loosening of the sac is of long duration and is the result of vigorous and ill-regulated taxis. This variety is rare. It was first described by Saviard, afterwards by Le Dran, Lafaye, and Richter. 3. Rupture of the neck of the sac and escape of the contents of the sac through the rupture into a cellular space outside of the peritoneum. The rupture generally occurs at the back of the sac just below the neck. This form of reduction has been well described by Birkett. If fluid alone escapes while the bowel remains, an illusory appearance of reduction will be presented. If in incomplete "false" reduction the rupture is at the distal extremity of the sac and the discharge of fluid subcutaneous, it is impossible, of course, for bowel to escape. As a variety of this form may be mentioned the passage of the strangled intestine through a cut made near the neck of the sac when a hernia knife had been introduced to divide the "stricture at the neck of the sac." Maydl has observed this accident. After the supposed division of the strangulating band, pressure is applied to the bowel, and reduction apparently occurs. The neck of the sac is, however, untouched, and the strangulation consequently persists. A very beautiful example of this kind is recorded by Farabœuf. This form also is rare.

A fourth form of "reduction in mass" is described and figured by Roser and mentioned by Pick. It consists in the reduction "into the inguinal canal" of the bowel that has formerly occupied the scrotum. A wide dilatation of the canal is considered as permitting this partial reposition. I have not been able to discover a museum specimen of such a condition, nor have I found any unequivocal case in the literature of this subject, though mention of the condition is made by several of the earlier writers on hernia. Chelius considered that in herniæ of moderate or large size, when reduction was apparently complete, a small segment of gut, thick with inflammatory products and paretic, might lie inert in the canal and undergo a later, secondary strangulation. Reduction of the strangulated contents of a hernial sac into a second underlying sac in the scrotum has been recorded by Armand, Horn, Sandyfort, and Scarpa. It is possible that these second sacs are pulled down into the scrotum by gubernacular bands, in the same manner as the sac of an infantile hernia. Demmeaux has suggested that whether the sac first present in the scrotum be congenital or acquired, the press of intestine on a weak and loosely supported neck may cause a diverticulum slowly to develop. Such pouch may be in front or behind, to the inner or outer side. If rupture of the neck of a femoral sac occurs the bowel may pass into the subperitoneal tissue. Callisen recorded in 1777 a case where, owing to rupture of the sac below the neck, the bowel had escaped and made for itself a lodgment between the pectineus and psoas muscles. These conditions may be represented in tabular form thus:—

Complete False Reduction.—1. Reduction into a properitoneal sac; rarely an interstitial sac. 2. Reduction of the whole sac with contents within the abdomen, outside the peritoneum, the mutual relations of sac and contents being undisturbed. 3. Rupture of the neck of the sac or its division during operation and extravasation of the hernial contents into the subperitoneal tissue.

Incomplete False Reduction.—1. Rupture of the sac at the neck or elsewhere with escape of fluid only. The hernial swelling becomes considerably smaller, but the bowel remains strangled. 2. Reduction of part of the hernial contents. A small portion remaining in the canal, paralysed and stiff with inflammatory products, undergoes a "secondary" strangulation.

If taxis prove ineffectual, operation must be resorted to. It is not possible to exaggerate the evils of delay, in cases of strangulation. There is nothing to be gained by delay, there is everything to be lost. Operation in itself is devoid of serious risk. Including all cases, the aged, infirm, or bronchitic, death would probably not occur in more than 5 per cent if operation were adopted at the earliest moment in cases of inguinal and femoral hernia. The mortality after strangulated hernia is considerably greater than that. During the last fourteen years at the Leeds infirmary, since a statistical report has been published, the mortality has been, for strangulated inguinal hernia, 17·4 per cent, and for femoral hernia, 23·8 per cent. The difference between these and 5 per cent is the *mortality of delay.* During the same period the operation for the radical cure has had a mortality in inguinal hernia of 2·3 per cent, in femoral hernia of 1 per cent. If the last five years only are selected the mortality is in both cases less than 1 per cent.

Treves gives the mortality of strangulated hernia as "over 30 per cent," Ross and Carless at "about 35 per cent." Now taxis, even if successful, so far as the reduction of the hernia is concerned, is not without its dangers. Bryant estimates the mortality after reduction by taxis in inguinal hernia 3·8 per cent, and in femoral hernia 10·5 per cent. It would probably be in

accord with the experience of most hospital surgeons to say that taxis applied for one or at the most two minutes is sufficient to reduce a hernia that can be with safety reduced. A longer application than this will produce such

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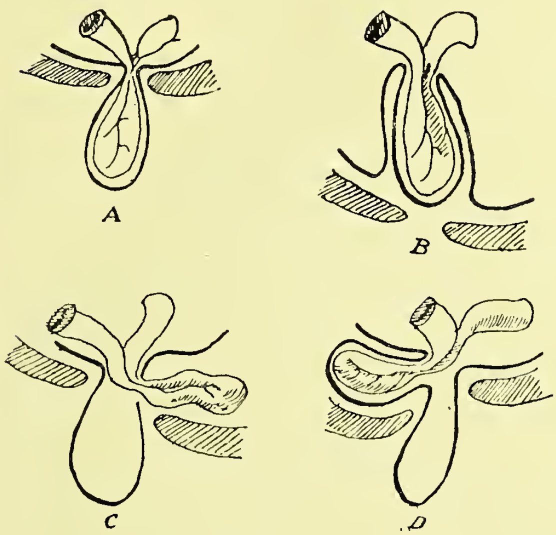

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Complete False Reduction.

FIG. 1.—A. Sac and contents in normal position.
FIG. 2.—B. Sac and contents displaced "en masse."
FIG. 3.—C. Rupture of sac near the neck.
FIG. 4.—D. Properitoneal sac.

damage in the gut as to render its reposition unsafe. When the patient is fully prepared for operation, and under the anæsthetic, a further brief attempt at reduction may be made, but failing then, the surgeon should proceed at once to operation.

Umbilical hernia, whether strangled or not, is a much more serious matter. The operation for the radical cure with us during the last fourteen years has had a mortality of 6·5 per cent, for strangulated hernia the mortality has been 56·4 per cent. The argument for early operation is therefore equally valid here.

Details of the Operation.—Immediately the hernial sac is opened the fluid contained therein should be emptied out and the sac with its contents washed gently but efficiently with hot sterile salt solution. The gut is then inspected.

A. If found to be living, the strangulating agent should be divided and the loop of bowel pulled further out of the abdomen, in order that the line of constriction may be examined. On no account should the finger be introduced into the neck of the sac with the idea of stretching the ring to permit more readily of replacement. If the deep groove which marks the point of constriction be not seriously damaged the bowel may, with gentle manipulation, be returned. If there is a line of ashen-grey slough this may be infolded by a layer of Lembert's or Halsted's sutures, and the gut replaced. Any omentum present may then be dealt with. Adhesions are often present, but it is not necessary to separate them, a process which is both slow and tedious. Traction on the omentum will bring the non-adherent part above the constriction into the sac; a series of ligatures is applied, and the omentum cut across below them. The neck of the sac is then ligatured and divided, and the sac and the adherent distal portion of the omentum are together stripped up and removed. A radical cure may be performed then by any of the usual methods.

B. If found to be in a doubtful condition the loop may be drawn, after division of the constriction, well down into the wound, and left there a few minutes for inspection. If there is any evidence of a gradual return of the natural colour the gut is still living; if there is none, and especially if the veins of the mesentery are felt to be blocked, the gut, if not dead, is certainly doomed. If, however, doubt still lingers, the gut may be returned just within the abdomen (where, owing to its paresis and the rapid formation of filmy adhesions, it will remain), and a tube passed down to it. Experience has abundantly shown that if the gut should give way its contents will in general pass along the hernial track and a fæcal fistula result. Or, and preferably, the loop of bowel may be drawn outside the wound, covered with a protective dressing, and left for 24 or 36 hours, then re-examined and dealt with according to its condition.

C. If found to be gangrenous two courses may be adopted.[1]

1. The strangled loop, with the implicated mesentery, may be excised, and the divided ends of the bowel stitched up, with or without a mechanical appliance.

2. The bowel may be opened with or without division of the constriction and an artificial anus formed. As modifications of this procedure may be mentioned the introduction of a Paul's tube after slitting open the bowel, or the stitching of the opened gut to the skin. In not a few cases where the gut has been laid open without division of the stricture the acute obstruction has continued. A careful division of the stricture without undue disturbance at the upper part of the sac can do no harm, and is therefore generally desirable.

Which of these two courses is to be adopted must depend upon many conditions to be carefully weighed by the surgeon. It must be admitted at once that *whenever possible resection should be performed.* A notable argument has

[1] The method of Helferich has been so seldom practised that its value is not yet determined. It consists in drawing out a loop of gut and uniting the ends by means of a Murphy button. The loop is then left, covered by an antiseptic dressing, and resection performed when the patient has rallied.

been made by many of the more ardent surgeons in a reference to statistics, showing that the mortality after resection is less than after the formation of an artificial anus. Thus in 394 cases of gangrene that I have tabulated, where an artificial anus was made, 30·7 per cent of patients recovered. In 443 cases of primary resection 53·9 per cent recovered, a difference of more than 20 per cent in favour of resection. Nothing could be more misleading than this argument. Resection is done in the less severe cases by surgeons adept and skilful in manipulation, with the most desirable surroundings and with adequate help. A surgeon who resected intestine in a moribund patient could have no sense of the fitness of things. In such a case the laying open of the gut would be performed to give the patient a last, slender chance; if death followed it would be due to the delay, not to the method. I hold the argument from statistics, therefore, as not valid, though it supports the method which is ideally the better. There are certain circumstances under which enterectomy, with stitching of the divided ends, could not be legitimately performed. These are—

(α) When the patient is profoundly collapsed from septic poisoning, almost pulseless and moribund; or aged, bronchitic, or otherwise in bad condition.

(β) Where prolongation of the operation would add a serious risk, as in cases where a large segment of gut is involved (as much as 6 feet has, however, been successfully removed).

(γ) Where strangulation has been of long duration. After 3-4 days' obstruction resection is rarely successful. In such cases the distension of the gut above the constriction is considerable, and the bowel does not lend itself readily to successful suture.

When, however, one meets with a case of recent, limited gangrene in a young, otherwise healthy subject, not exhausted by vomiting nor collapsed by the absorption of poisonous material, resection will find its most successful application.

The disadvantages of the method in which an artificial anus are formed are neither few nor inconsiderable. Briefly they are:—

(α) The possibility of the continuance of symptoms of acute obstruction, a possibility by no means remote, if the stricture is not adequately divided.

(β) Septic infection from the wound in the gut or from the sac spreading to the peritoneum, or infecting a wide area surrounding the opening.

(γ) The opening in the gut may be high up in the small intestine, and inanition may ensue.

(δ) The necessity of a second operation, which is not without its own mortality.

We are not, however, in a position to say that the application of either method should be unvarying. Each has its uses. No hard and fast rule can be made; the surgeon can only be guided by the general principles laid down; the decision he makes will depend upon his own resourcefulness, and his readiness and capacity to appreciate the relative value of many signs.

Complications of Strangulated Hernia.—Among the more important of these may be mentioned—

1. *The persistence of symptoms after reduction*, due to—

(*a*) False Reduction, complete or incomplete (*see* page 191).

(*b*) Acute enteritis in the snared loop leading to paralysis, and going on perhaps to ulceration, gangrene, or perforation.

(*c*) Acute peritonitis from infection by the damaged gut or by the escape of infected fluid from the sac.

(*d*) The existence of acute strangulation, apart from the hernial protrusion, or connected with it.

(*e*) The existence of another hernia, femoral, obturator, etc., which is strangled.

(*f*) The existence of a condition where symptoms are mistaken for strangulation in a hernia, *e.g.* lead poisoning.

If (*b*) or (*c*) are present the abdomen should be opened; if the former condition is found an artificial anus should be made; if the latter, the cavity may be flushed and drained.

2. *Affections of the Lungs.*—Pneumonia and bronchitis are seen, not infrequently, in cases of strangulated hernia both before and after reduction. Verneuil, in 1881, called attention to an intense pulmonary hyperæmia as a factor in causing death. Gussenbauer regarded the condition of the lungs as the result of infective embolism from the mesenteric vessels; Lesshaft suggested that infection was probably conveyed by inhaled particles of putrid vomit, especially during anæsthesia.

The signs of pulmonary trouble do not, as a rule, become manifest till 12, 24, or 36 hours after reduction. There is at the first some respiratory concern, hardly amounting to difficulty, a little hurry in the rate of breathing, a trifling elevation of temperature. In a few hours all these conditions are accentuated, and pneumonia, usually of a very aggressive type, is developed. The investigations into the bacteriology of strangulated hernia are of interest in this connection (*see* page 190).

As more or less remote sequelæ of strangulated hernia should be mentioned *stricture of the intestine in the strangled loop, and acute intestinal obstruction from the formation of peritoneal adhesions.*

Inguinal Hernia

Inguinal Hernia is by far the most common form of hernia met with. According to Macready, in 100 men ruptured, 97·5 per cent have inguinal

and 2·5 per cent femoral hernia. Among 100 women ruptured, 60·3 per cent have inguinal and 39·7 per cent femoral hernia. Among 100 ruptured persons of both sexes the following proportions obtain:—Male inguinal, 83·5 per cent; female inguinal, 8·5 per cent; female femoral, 5·9 per cent; male femoral, 2·1 per cent.

Anatomy of Inguinal Hernia.—An inguinal hernia leaves the abdomen by the inguinal canal, entering at the internal abdominal ring and emerging at the external. The canal is $1\frac{1}{2}$ in. long in the adult. The anterior wall is formed by the aponeurosis of the external oblique, and in the outer third by the fibres of the internal oblique arising from Poupart's ligament.

The posterior wall is generally described as being formed in its inner two-thirds only by the conjoined tendon, but this statement needs correction. The conjoined tendon forms the inner part of the posterior wall, but in the outer part there is, in the great majority of cases, a quite distinct layer of tendinous fibres derived from the transversalis muscle. This layer (sometimes referred to as the "reflected tendon" of Cooper) extends up to, and forms, the inner boundary of the internal abdominal ring, where there is a noticeable thickening, the "ligament of Hesselbach" descending from the outer limb of the fold of Douglas.

The posterior wall is also formed by the triangular fascia of Colles, sometimes referred to as the "posterior" pillar of the external abdominal ring, and the transversalis fascia, here thickened by the addition of bundles of oblique and transverse fibres. The conjoined tendon is a very variable structure. In the great majority of cases it is thin and weak, and blends internally with the sheath of the rectus.

The floor of the canal is formed by Poupart's and Gimbernat's ligaments, and the roof by the arching fibres of the internal oblique and transversalis.

Action of the Inguinal Canal.—The term "canal" is perhaps unfortunate if it conveys the idea of an open tube transmitting the cord, for nothing of the kind exists when the conditions are normal. In a healthy individual the anterior and posterior walls are in close apposition. When the arched fibres of the internal oblique contract, they become straight and descend towards Poupart's ligament, compressing the spermatic cord against that structure. While so descended they are gripped and fixed by the pressure of the anterior and posterior walls, which, by the contraction of the external oblique and the transversalis, are made tense and are approximated. The canal therefore acts as a "sphincter," and would, with greater accuracy, be described as the *Inguinal valve.* For the efficient working of this valve it is necessary that the "canal" should be empty. Any structure, such as a deposit of fat along the cord, or a pleated sac after operation, which occupies the canal, will prevent the descent of the arched fibres of the internal oblique and transversalis, and thereby make impossible the normal sphincteric or valvular action of the canal.

If the lower portion of the anterior abdominal wall is examined from behind certain elevations with intervening depressions will be noticed. Along the middle line a fold, the *plica umbilicalis media*, extends from the bladder along the urachus to the umbilicus; on each side of this there is a ridge, the *plica umbilicalis lateralis*, raised up by the obliterated hypogastric artery, and still further outwards a slighter fold, the *plica epigastrica*, indicating the line of the deep epigastric artery. Between the two first folds is a recess, the *fossa supra vesicalis*, between the second and third the *fovea inguinalis mesialis*, and to the outer side of the plica epigastrica the *fovea inguinalis lateralis.* Occasionally the line of the obliterated hypogastric coincides with that of the deep epigastric, and only a single ridge is raised up.

The plica epigastrica, as it passes upwards and inwards, forms, with the outer edge of the rectus to the inner side and Poupart's ligament below, a triangular interval known as the *Triangle of Hesselbach.*

An oblique inguinal hernia descends, outside the plica epigastrica, through the fovea inguinalis lateralis. A direct inguinal hernia may pass (*a*) between the plica epigastrica and the plica umbilicalis lateralis, or (*b*) between the latter and the central fold; the former is known as a *superior* or *external*, and the latter as an *inferior* or *internal* direct inguinal hernia.

The coverings of an oblique inguinal hernia are skin, superficial fascia, intercolumnar fascia, cremasteric fascia, infundibuliform fascia, subperitoneal tissue, and peritoneum. Of a superior or external direct hernia, skin, subcutaneous tissue, intercolumnar fascia, cremasteric fascia, and some fibres derived from the tendon of the transversalis muscle at or near the ligament of Hesselbach, transversalis fascia, subperitoneal tissue, and peritoneum. Of an inferior or internal direct hernia, skin, subcutaneous tissue, intercolumnar fascia, Colles' triangular fascia, conjoined or transversalis tendon, transversalis fascia, subperitoneal tissue, and peritoneum.

The following varieties of inguinal hernia will be described:—

Oblique Inguinal—

Skin.
Superficial fascia.
Intercolumnar fascia.
Cremasteric fascia.
Infundibuliform fascia.
Subperitoneal tissue.
Peritoneum.

Superior Direct—
- Skin.
- Superficial fascia
- Intercolumnar fascia.
- Cremasteric fascia.
- Transversalis tendon.
- Transversalis fascia.
- Subperitoneal tissue.
- Peritoneum.

Inferior Direct—
- Skin.
- Superficial fascia.
- Intercolumnar fascia.
- Colles' fascia.
- Conjoined or Transversalis tendon.
- Transversalis fascia.
- Subperitoneal tissue.
- Peritoneum.

(A) Oblique Inguinal Hernia.

I. The acquired form.

II. Hernia depending upon congenital anomalies in the processus vaginalis.

(*a*) Congenital Hernia; (*b*) Hernia into the funicular process; (*c*) Infantile Hernia; (*d*) Encysted Hernia; (*e*) Hernia with retention of the testis; (*f*) Hernia into the canal of Nuck.

III. Properitoneal Hernia.

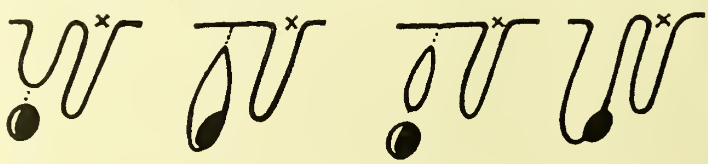
IV. Interstitial Hernia.

(B) Direct Inguinal Hernia.

A. Oblique Inguinal Hernia

I. *Acquired Inguinal Hernia.*—This form of hernia generally makes its first appearance in adult life. When confined to the inguinal canal the swelling is known as a bubonocele; after escape from the canal it becomes a *scrotal* or *complete* hernia. The sac is formed slowly and is derived from the parietal peritoneum. When the hernia reaches the external abdominal ring, it is free to enlarge in all directions. The result is that a globular scrotal swelling is connected to the abdomen by a narrow neck; the neck being the portion lying in the canal. As the tumour enlarges, the neck (as a result of the approximation of the internal to the external abdominal ring from traction exerted by the hernial mass) becomes progressively shorter. The scrotal portion descends down to, and enlarges in front of, the testis. The sac is never intimately adherent to the structures of the cord.

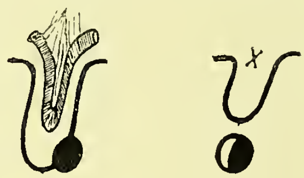
II. *Hernia depending upon congenital anomalies in the processus vaginalis.*—These forms are probably much more common than is generally believed. There may be little or nothing to distinguish an ordinary (so-called acquired) hernia from a hernia into the funicular process, and Russell has recently suggested that all herniæ are congenital in the sense that they occur in preformed sacs derived from the processus vaginalis. It is certainly quite common to find in adult bodies a fine funicular process unclosed when there has never been a history of any hernial descent. If the upper portion of this process remained open and formed a hernial sac, the case would almost certainly be looked upon as acquired, though in reality dependent upon congenital abnormality in the processus vaginalis.



C 1 C 2 C 3 C 4

Fig. 7.—Infantile hernia.—4 varieties.

(*a*) *Congenital Hernia* is characterised by the absence in the processus vaginalis of any attempt at closure. The process remains open throughout and forms a ready-made hernial sac into which the gut descends without hindrance. In the sac the hernial content is in touch with the testis. A hernia of this form frequently appears quite suddenly, and in a young adult is not uncommonly strangulated at its first emergence. Constrictions, the result of attempted closure at certain points, may give rise to an "hour-glass" sac. The most common narrowing of this kind is situate at the upper end of the tunica vaginalis.



A B

Fig. 5.—A. Congenital hernia.
Fig. 6.—B. Hernia into the funicular process.

(*b*) *Hernia into the funicular process* results when the tunica vaginalis is fashioned from the lower part of the processus, while the upper part remains unchanged. This hernia possibly forms the majority of the cases of inguinal hernia.

(*c*) *Infantile Hernia.*—The term "infantile" has no reference to the period at which the hernial tumour first appears. The admirable work of Lockwood in this matter has been generally accepted by all subsequent writers. This author first described four varieties of infantile hernia. In each of them a second peritoneal process is pulled down into the scrotum behind the processus vaginalis, by an aberrant attachment of the gubernaculum. The varieties of infantile hernia depend not upon the condition of this second peritoneal tube, but upon the varying states of closure in the processus vaginalis, according as to whether this process is (1) closed below and open above; (2) closed above and open below; (3) closed above and below; (4) open throughout. I have seen one example of an infantile sac, closed at its upper end, containing hydrocele fluid. In the un-

closed processus vaginalis a congenital hernia had formed.

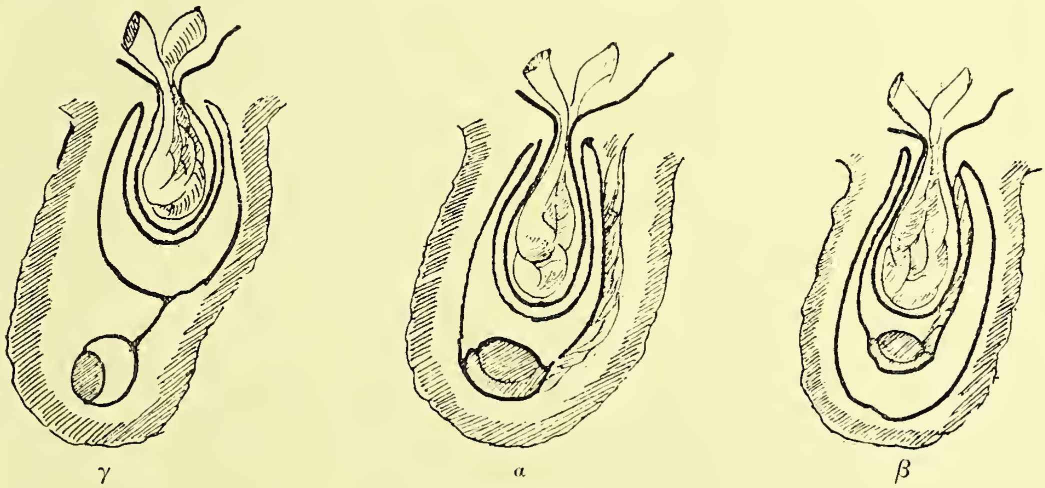
(*d*) *Encysted Hernia.*—I described in my "Arris and Gale" Lectures three forms of encysted hernia—

α. Encysted hernia with the testis lying at the bottom of the sac.

β. Encysted hernia with the testis lying at the apex of the hernia.

γ. Encysted hernia of the funicular process.

α. This form depends upon the closure of the vaginal process only at the internal abdominal ring. A hernia now develops, the sac being formed from the parietal peritoneum,



Figs. 8, 9, 10.—Encysted hernia.

and as it descends reaches the upper end of the large tunica vaginalis. This it dimples and then, in its steady increase, invaginates until the hernial sac hangs pendent from the internal abdominal ring, in the cavity of the tunica vaginalis.

β. This form is similar to α, except that it is associated with retention of the testis in the inguinal canal. The hernial sac in its descent meets with the testis, which it carries before it and invaginates into the tunica vaginalis.

γ. This form is the same as α, except that the tunica vaginalis is shut off and the hernia is encysted in the funicular process.

(*e*) *Hernia with retention of the testis* is not unusual. The testis as a rule is retained in the inguinal canal or at the external abdominal ring. The inguinal valve is prevented from acting, and the descent of a hernia is thereby invited. The testis is generally small and soft, almost always atrophied, and is at times exceedingly sensitive. Cases of hernia with retention of the testis may be treated by the application of a truss, the testis being disregarded, but are more satisfactorily dealt with by castration and the performance of a radical cure.

(*f*) *Hernia into the canal of Nuck.*—If the process of peritoneum accompanying the round ligament in the female remain unclosed, it forms the "canal of Nuck." A hernia into this process corresponds to a congenital hernia in the male.

III. A *Properitoneal Hernia* fulfils the following conditions—

i. The hernial sac has two loculi.

ii. The inner loculus lies between the peritoneum and the fascia transversalis.

iii. The outer loculus lies in the inguinal (or crural) canal, or in rare cases between the layers of the abdominal wall.

iv. Both loculi open into the abdomen by a single orifice, the "ostium abdominale."

The inner loculus may occupy one of three positions—

1. It may pass upwards and outwards towards the anterior superior spine.

2. It may pass directly backwards and occupy the inner part of the iliac fossa.

3. It may pass downwards and inwards into the true pelvis and lie by the side of the bladder.

The right side is more commonly affected than the left. So far as causation is concerned little is accurately known. The evidence may be thus summed up. The hernia arises most frequently as the result of some congenital abnormality in the inguinal region—as an added factor there may be a loosening (congenital in origin, Linhart) of the parietal peritoneum. In some cases a more or less complete "reduction in mass" may be the responsible factor. In a few cases the inner sac may be first formed, and the inguinal sac be a secondary diverticulum. (For further details see the report of my lectures, *Lancet*, Feb. 24, 1900.) The peritoneal conditions are similar to those found in "bilocular hydrocele."

IV. An *Interstitial Hernia* fulfils the following conditions—

1. The hernial sac has two loculi.

2. The inner, upper loculus lies (*a*) between

the internal and external oblique (interparietal), or (*b*) between the external oblique and the skin (extra-parietal).

3. The outer, lower loculus lies in the inguinal canal.

4. The upper loculus opens into the lower in some cases close to, in other cases a little distance from the internal abdominal ring.

In 73·4 per cent of cases in males there is some abnormality of the testis. The hernia is, however, of decidedly greater relative frequency in females than in males. Macready gives ·13 per cent of all cases in males and ·61 per cent in females.

The development of the interstitial sac in males depends almost certainly upon the incomplete testicular descent. The testis bars the way to a further protrusion of the hernia, and the inguinal sac therefore bulges out in the direction of least resistance. This explanation is strengthened by the observation, which is almost universal, that the scrotum is hardly ever fully occupied by a sac containing bowel or omentum. In 129 cases observed at the Truss Society, in only 9 was the scrotum occupied by the hernial swelling. Macready attributes the origin of this form of hernia to a "wasting or congenital defect" of the muscles below the level of the anterior superior spine.

B. Direct Inguinal Hernia

Direct Inguinal Hernia is always acquired. It is in reality a ventral hernia occurring at the semilunar line. The conjoined tendon, or the tendon of the transversalis, may be split, or, more usually, carried in front of the hernial mass. Direct hernia is the hernia of middle age. A hernia appearing for the first time in a man of fifty years of age or over is probably of the direct variety. Among 63 single direct hernia 14 were scrotal and 49 in the canal; and among 61 double direct, 36 were bubonoceles, 5 were scrotal, and in 20 the older hernia was scrotal, and the recent one bubonocele (Macready).

Signs and Symptoms of Inguinal Hernia.—The general signs and symptoms of reducible and irreducible herniæ have been already set forth, and it only remains to emphasise the special features occurring in examples of inguinal rupture. In the earliest stage a mere bulging at the internal ring may be observed during expulsive efforts. By degrees the canal fills up until the external ring is reached, when the hernial swelling tends to increase rather more swiftly and to become globular in form. In a direct hernia there is no obliquity of the neck, but the mass projects directly outwards. On examining with the index finger invaginated through the external abdominal ring there is a striking difference in the two forms of rupture. In oblique hernia the finger passes upwards and outwards along the canal and perceives the arching fibres of the internal oblique and transversalis. In the direct form the finger passes at once backwards into the abdomen. A congenital hernia may at its first appearance traverse the whole length of the processus vaginalis and reach the testis. A hernia into the funicular process may immediately descend well into the scrotum; if so, it will be cylindrical rather than globular in form. An infantile hernia has no special clinical features.

A properitoneal hernia is generally first recognised when symptoms of acute or subacute strangulation develop. In those cases where the diverticular sac projects outwards or backwards a swelling may be noticed. In 22 cases out of 36 recorded by Breiter, a "tumour or tumefaction" was noticed in 22. This proportion is probably in excess of the truth, for many cases of properitoneal hernia are recognised only as examples of "reduction in mass."

In interstitial hernia the mural sac is always capable of being recognised. At one time or another the sac can be seen distended with the usual hernial contents, and no difficulty will be experienced in the recognition. The symptoms are inconspicuous, and strangulation is infrequent.

Differential Diagnosis.—From *Femoral Hernia.*—The difficulty in distinguishing inguinal from femoral hernia is far greater in women, especially stout women with indefinite landmarks and small ruptures, than in men. The crease in the skin of the groin is at its inner end, very close to the pubic spine. If this point be found and the finger placed upon it, the hernia above and internal to the finger is inguinal, and below and external to it femoral. If the hernia has reached the labium or the scrotum it must, of course, be inguinal. If the hernia be reducible, the point of its emergence from the internal abdominal or femoral ring may be ascertained.

From *Scrotal Swellings.*—The chief faults in the diagnosis of inguinal hernia are concerned with swellings of the scrotum, hydrocele in its various forms, enlargements of the testis, varicocele, hæmatocele and hydrocele of the cord. In examining a tumour occupying the scrotum it is necessary, in the first place, to determine whether the tumour is primarily scrotal or primarily abdominal, and an attempt must therefore be made to define the upper end of the swelling. In all primary scrotal swellings except infantile hydrocele, the upper limit of the tumour can be determined and the cord felt there. In infantile hydrocele the processus vaginalis is closed only at the internal abdominal ring, and fluid accumulates below this point.

A *vaginal hydrocele* is a scrotal tumour, generally rounded in outline, fluctuating and translucent. There is no impulse on coughing. If the early history can be obtained, it will be found that the swelling began at the testis and not at the inguinal canal.

A *congenital hydrocele* is translucent, fluctuating, and reducible. The reduction is slow and even, there is neither slip nor gurgle. The tumour returns gradually, and the bottom of the scrotum is first occupied.

A *hydrocele of a hernial sac* has been already described. It presents the same features as a vaginal hydrocele, but may have a thick, solid neck.

A *hydrocele of the cord* is rounded and translucent, and feels like a grape slipping along the cord. It may be reducible, and descends upon coughing. Traction on the testis generally communicates a slight movement to the hydrocele.

A *varicocele* is reducible, reappears on standing, and may have a decided impulse on coughing. The impulse, however, gives no shock to the hand, but is merely the result of the turgid condition of the veins. Reappearance of the swelling is prevented only by forcible pressure along the canal when the patient is standing; a gentle pressure sufficient to retain the bowel or omentum permits the reappearance. The irregular knotted "worm-like" feel of the swelling may simulate omentum fairly closely.

The solid tumours of the testis present little difficulty in diagnosis, and need not be further considered.

From *labial swellings*.—A hernia of the labium is practically always reducible. Cysts of the labium and hydroceles of the canal of Nuck are irreducible, smooth, translucent, and fluctuating. Varices of the labial veins may, when large, resemble hernia, but a dilatation of the surface veins will generally suggest the correct diagnosis.

Inguinal Hernia in Children

Inguinal hernia is very commonly seen in children of both sexes, forming in males 23·4 per cent, and in females 22·5 per cent of all cases. It is more frequent upon the right side, and descends almost invariably in a partially or completely unclosed vaginal process. The diagnosis of the condition is rarely a matter of doubt or difficulty.

The most active of the determining *causes* is ill-feeding, leading to gaseous distension of the intestine and increased intra-abdominal pressure; others of less influence are phimosis with adhesion of the prepuce to the glans and retention of the secretion of Tyson's glands, vesical calculus, rectal polypus, intestinal irritation by parasites, or any condition leading to persistent straining on the part of the patient.

Treatment. — The very great majority of patients can be satisfactorily treated by trusses. The "wool-truss" has been much vaunted as an efficient, simple, inexpensive instrument. Personally I have been most dissatisfied with it, and now have abandoned it entirely. The best form of truss is a "spring truss," covered with india-rubber of the best quality. Attention must be paid to the proper fitting and adjustment of the truss, and to cleanliness and dryness of the skin. The truss may only be removed for purposes of cleanliness. As to the length of time for which the truss must be worn, Mr. Langton, whose experience is unrivalled, gives the following instructions:—"When the protrusion takes place before the age of 1, the use of the truss should not be discarded under any circumstances till the age of 4 years; if a truss has not been worn till the age of 3 or 4, it must be worn till the age of 10; if not worn till the age of 7, then the truss should be worn till puberty."

Operation is rarely called for. It should only be advised—

1. In cases of irreducible omentum.
2. In all cases where fluid is present in the sac.
3. In operations for the relief of strangulated hernia.
4. In all cases where it is impossible to return and control the hernia by mechanical appliances.
5. In cases where a truss has been worn for 3 or 4 years without benefit.

Strangulated Hernia in Children.—Strangulated hernia requiring operation during infancy is a very unusual occurrence. The most recent contributions to the literature of the subject have been published by Carl Stern and by Tariel of Paris. The extreme rarity of the condition is appreciated by both writers. Stern, in order to determine its frequency, consulted the records of the children's hospitals of Basle, Prague, Breslau, Vienna, Krakow, Frankfort, Amsterdam, Berne, and Göttingen.

In these hospitals for four consecutive years 139,000 children were treated, but there is no record of any case of herniotomy for strangulation. Of 1900 cases operated upon for strangulated hernia in various hospitals, 13 occurred in children. The proportion of cases in adults as compared with children is calculated by Stern to be in the ratio approximately of 108 to 1. Tariel, after remarking that several noted surgeons, among whom are Holmes, Gosselin, St. Germain, and Lannelongue, have never met with acute cases requiring herniotomy, tabulates the records that he has been able to collect. They number in all 128. König states that throughout his long surgical career he has only met with two cases requiring operation in the "early years" of childhood. Nussbaum operated upon two cases among a total number of 54,000 children under his care. Broca found strangulation in 9 cases out of 200 requiring radical cure in infancy. The first collection of records of operations of this kind was made by Ravoth, who tabulated 30 well-authenticated cases. Fére, who followed him, investigated the records of 52 operations in 56 cases. Howard Marsh in 1874 collected 47 cases. Knobloch, whose investigations preceded those of Stern,

compiled notes of 87 herniotomies for inguinal, 11 for umbilical, and 1 for femoral hernia. To this already comprehensive list Stern added 51 later cases of inguinal and 3 of umbilical hernia.

It is remarkable that operations are more frequently called for during the earlier months of childhood than during the later. The greatest number occur in the first, second, and third months of the first year. The proportion after this period diminishes very rapidly, as is shown by comparing in a tabular form the cases operated upon in each of the first twelve months.

During the	1st month		16	cases occurred.
,,	2nd	,,	15	,,
,,	3rd	,,	14	,,
,,	4th	,,	9	,,
,,	5th	,,	4	,,
,,	6th	,,	7	,,
,,	7th	,,	3	,,
,,	8th	,,	6	,,
,,	9th	,,	9	,,
,,	10th	,,	3	,,
,,	11th	,,	3	,,
,,	12th	,,	4	,,

The symptoms and the signs are similar to those already described as being present in adults. In nearly all the recorded cases retention of urine has been observed.

Treatment.—If careful taxis prove unavailing, resort will be had to operation, which is carried out as in adults. In young children a "radical cure" is generally effected by the simple removal of the sac.

Sudden death after operation without evident or sufficient cause has been noticed by several surgeons.

Femoral Hernia

Femoral or Crural Hernia (merocele) escapes from the abdomen at the crural ring, passes down the crural canal and emerges upon the thigh through the saphenous opening. It is only very rarely congenital in origin.

The crural ring is marked on the abdominal aspect by a slight depression, the *fovea femoralis*, and is closed by a specialised portion of the subperitoneal tissue, containing a lymphatic gland, known as the "septum crurale" of Cloquet. The ring is bounded *in front* by the superficial and deep crural arches, *behind* by the pubic bone covered by the pectineus and the pectineal fascia, to the *outer side* by the inner partition of the crural sheath which separates the ring from the external iliac vein, to the inner side by Gimbernat's ligament and the other fibrous structures attached to the ilio-pectineal line.

The epigastric artery is placed at the upper and outer portion of the ring, and the pubic branch of this vessel runs transversely inwards to Gimbernat's ligament. In about 30 per cent of cases the obturator artery arises from the epigastric and may then pass down on the outer side of the ring (54 per cent), directly across the ring (37 per cent), or on the front and inner side of the ring (9 per cent).

The upper part of the pectineal fascia is considerably strengthened by a tough white band of dense fibrous tissue running along the ilio-pectineal line; some of these fibres are continued into Gimbernat's ligament, and others pass across the middle line and meet those of the opposite side. This band is known as *Cooper's ligament*, or the ligament of the pubis.

The crural canal is about half an inch in length. In front of it the upper margin of the saphenous opening arches inwards to join Poupart's and Gimbernat's ligaments. That portion of the upper edge lying internal to the femoral vein is known as Hey's ligament or the femoral ligament.

A hernia passing along the canal descends at first vertically, but on reaching the saphenous opening it bulges forwards, and afterwards tends to enlarge in an upward and outward direction.

The coverings of a femoral hernia are skin, superficial fascia, cribriform fascia, the femoral sheath, the septum crurale and subperitoneal tissue, and the peritoneum. The femoral sheath and the septum crurale are generally blended into one firm layer described by Cooper as the "fascia propria."

Femoral hernia is more common in the female than the male, probably because the pelvis is proportionately wider and the crural ring actually larger than in the male. The symptoms of the rupture are similar to those present in cases of inguinal hernia, but are of a distinctly less aggressive type. A femoral hernia may pass quite unnoticed, especially in stout women, and indeed the abdomen may be opened upon a diagnosis of acute obstruetion when the symptoms are due to a strangulated femoral partial enterocele of inconspicuous size.

Differential Diagnosis.—The diagnosis of femoral from inguinal hernia has already been discussed. The other conditions simulating femoral hernia are—

Adenitis.—A single enlarged femoral gland placed over the saphenous opening may very closely simulate an irreducible epiplocele. Such a gland is superficial, movable from side to side, and has no deep attachment; in all these points differing from a rupture.

Varix of the Saphena Vein.—A varix oecupies almost exactly the position of a femoral hernia, has an indistinct impulse on coughing, increases in size upon standing, and is reducible. The venous distension will, however, reappear on standing if pressure be kept up on the crural canal. As a general rule, there are other dilatations of the veins of the leg and thigh on the same side.

Hydrocele of a hernial sac.—This may very closely simulate a hernia, but the fluctuation and possible translucence are distinguishing

features. A distension of the *ilio-psoas bursa* may form a swelling resembling very closely in contour and feel, but differing in position from, a hernial protrusion.

Psoas abscess appears outside the femoral vessels. There is an impulse on coughing, and fluctuation between the iliac and femoral portions can generally be elicited.

Hypertrophy of the subperitoneal fat may result in the presence of a tumour in the crural canal and at the saphenous opening; the down growth of this mass may drag into the canal a small protrusion of peritoneum. The condition is physically identical with an irreducible femoral hernia, but the history generally shows that the tumour has been very slow in growth and, from the earliest beginnings, irreducible.

The following rare varieties of femoral hernia have been described:—

1. *Cloquet's Hernia* (pectineal hernia), where the hernia lies beneath the pectineal fascia, on the pectineus muscle.

2. *Laugier's Hernia*, where the sac is protruded through an aperture in Gimbernat's ligament.

3. *Hesselbach's Hernia*, where a series of diverticula are sent off from the sac through the openings in the cribriform fascia. This form was described in 1814 by F. C. Hesselbach.

4. *Cooper's Hernia*, where a series of diverticula are sent off from the sac through openings in the superficial fascia.

5. *External Femoral Hernia*, where the sac descends between the femoral artery and the anterior superior spine. This form was first described by A. K. Hesselbach in 1829, but is often incorrectly described as Partridge's hernia.

Cruro-properitoneal Hernia is decidedly rare. The additional sac passes inwards towards the pelvis. Cases of "reduction in mass" have been recorded by Farabœuf and others.

Umbilical Hernia

Umbilical Hernia is of three varieties—

1. Congenital Hernia.
2. The Hernia of Infants.
3. The Hernia of Adults.

1. *Congenital Hernia.*—At the beginning of the third month of intra-uterine life the viscera becomes enclosed in the abdominal cavity by the growth of the visceral plates. If this growth is defective, the visceral enclosure is more or less incomplete and a "hernia" results. The term "hernia," as Malgaigne suggests, is inappropriate, "for we are not concerned with viscera escaped from a cavity, but with viscera which have never entered it."

Three classes of this hernia are described—

(*a*) Where the gap at the umbilicus is small; hernia of the root of the cord.

(*b*) Where the gap is moderate in size; sacculated hernia.

(*c*) Where the abdominal wall is grossly incomplete (eventration); the viscera having a thin covering derived from the amnion.

In group (*a*) the pressure of a pad and bandage will generally effect a cure. In group (*b*) operation is necessary. The cases in group (*c*) are of merely pathological interest.

2. *The Umbilical Hernia of Infants.*—This form arises after closure of the abdominal wall, and is due to a yielding of the umbilical cicatrix. It is very frequent in both sexes, appears some weeks or months after birth, and is probably due to intestinal disorder. Almost without exception the cases are spontaneously cured. It is only necessary to apply a large firm pad and bandage to prevent protrusion of the hernia when the infant cries or strains.

3. *The Umbilical Hernia of Adults.*—This form of hernia develops comparatively late in life, and is not concerned with the variety just described. It is exceedingly rare for an infantile umbilical hernia to remain until adult age.

If the anterior abdominal wall be examined from behind, and the peritoneum be removed, a local thickening of the transversalis fascia in the neighbourhood of the umbilicus will be observed. Interlacing bundles of horizontal fibres form here a distinct layer known as the "fascia umbilicalis" or the "fascia of Richet." The extent of this fascia and its strength vary within considerable limits. Sachs, who has investigated this matter with conspicuous ability, describes three varieties as occurring:—

(1) A dense fascia most strongly developed behind the umbilicus, and thinning off above and below.

(2) A fascia of similar character so far as the upper portion is concerned, but with a sharply-defined concave margin below. The level of this margin may be lower than, or opposite to, the umbilicus.

(3) A thinner fascia situated entirely above the umbilicus.

Between the fascia of Richet and the linea alba is a passage, the "*canal of Richet*," which contains fat, the ligamentum teres and four or five small para-umbilical veins. The entrance to the canal is directed upwards.

In the second and third varieties of the fascia it is occasionally noticed that small peritoneal diverticula are present at the lower sharply-defined concave margin. These pouches may be quite inconspicuous, or they may pass outwards through the umbilicus. Malgaigne and others have looked upon these diverticula as congenital in origin, but according to Sachs, whose observations I have verified, they are never seen before the second month of extra-uterine life. When present they may be considered as predisposing to the onset of hernia.

The lower portion of the umbilical scar has been shown by Herzog to be denser and stronger than the upper. The umbilical arteries are said

to be enveloped in a firmer and more fibrous sheathing than the vein, and when the cicatricial obliterative process ensues, the scar formed around them is correspondingly more resistant and tougher than that around the thin-walled vein.

Kocher has described two forms of hernia occurring at the umbilicus in adult life: an *oblique*, which descends along the canal of Richet, lies between the fascia of Richet and the linea alba, and escapes at the navel; and a *direct*, which passes immediately outwards at the level of the umbilicus. So far as I am aware, the only author who has verified Kocher's description of the oblique form is Jaboulay, and that only in a single case. It was taught by Petit, Scarpa, and many other surgeons, that an umbilical hernia did not escape *at* the umbilicus, but through an adjacent opening, either above or below, but more commonly above. It is not improbable that some of the examples of so-called "umbilical" hernia are in reality cases of ventral hernia through the linea alba (adombilical).

Signs and Symptoms.—This form of rupture is usually the prerogative of the corpulent. Women, especially those who have borne children, are affected far more frequently than men.

In 775 cases collected by Macready, 566 occurred in women, and 209 in men.

The rupture appears very gradually, and may for a time pass unnoticed; it forms at first a smooth, globular, painless swelling. With increasing size it becomes irregular in shape; sulci appear and divide the mass into lobules of varied size. Such depressions are due to adhesions of the contents to the sac wall. The coverings of the hernia are always thin, but increasing distension from within and the chafing of clothes upon the surface may cause them to ulcerate and very occasionally to give way. The swelling, when large, is pendulous and pedunculated. The umbilical scar, stretched and considerably altered in appearance, is seen usually upon the lower half of the swelling. A rupture of even moderate duration is almost without exception irreducible. The omentum is always a content of the sac and generally lies anteriorly; this fact accounts for the lobulated irregular feel that most herniæ possess, and for the frequency of the irreducibility. The *symptoms* of an umbilical hernia are, as Sir Astley Cooper first said, more pronounced than in any other common variety of rupture. Pain, flatulence, vomiting, and intestinal irregularity are certainly more frequent, and tenderness of the hernia is decidedly more pronounced. Incarceration is oftener seen than in any other rupture. Strangulation does occur, but not so frequently as is generally supposed; when present, it is generally the outcome of, and the final event in, a somewhat prolonged obstruction.

Treatment.—In the early stages, when the hernia is small and perhaps reducible, the best support is obtained from a broad, sufficiently strong, and well-made abdominal belt with a large round shield to cover the umbilical area. If the hernia is of old standing, large and irreducible, a truss with a pad of chamois leather will be needed. The circumference of the bag must consist of an oval plate, and the bag should be made of a depth rather less than that of the hernial bulging, so that a very slight degree of pressure may constantly be exerted upon the rupture. In addition, careful attention must be paid to the diet, and the patient, if inclined to obesity, should endeavour to effect a reduction in the body weight.

But an umbilical hernia, however rigidly cared for, is a constant source of danger, and therefore, whenever possible, and whenever safe, an operation should be advised. The general condition of those who suffer from this form of hernia, however, is not as a rule very satisfactory. They are persons of indolent habit, loose-bodied and flabby, and they do not readily brook surgical intervention. If, however, there is no definite hindrance, it is probable that there is more security in operation than in the treatment by trusses.

Umbilical herniæ are especially apt to become incarcerated, and the mortality of operation in times of stress is, as we have seen, considerable. The cases for operation must of course be selected with considerable care, but when so selected there is no reason to suppose that the mortality is unusually large.

Operation.—If the swelling be large, two incisions enclosing an ellipse of skin will be made, and the superfluous skin removed. On opening the sac the bowel will be returned and the omentum removed. In order to effect a satisfactory and permanent closure of the opening, it is advisable to stitch the abdominal layers up separately.

This procedure I generally carry out in the following manner. The peritoneum is first stitched up with a continuous layer of catgut. The inner rounded margin of the rectus sheath on each side is then divided for the whole length of the wound by a vertical incision midway between the anterior and posterior surfaces, until the muscular fibres are reached. Then the posterior part of the sheath is stitched to its fellow across the middle line by a series of interrupted silkworm-gut sutures. Similarly the muscle-fibres of the two recti are stitched, and afterwards the anterior layers of the sheaths. Finally, the skin and subcutaneous tissues are closed in the usual manner. There are thus five layers of sutures, and four of the layers are buried. For the buried stitches I prefer silkworm-gut, which can be effectively sterilised, but kangaroo tendon or strong chromicised catgut answers very well.

There can be no doubt that recurrence after operation is more frequent in umbilical hernia than in inguinal or femoral; and for this reason it is always a wise precaution to insist upon the subsequent wearing of a stoutly-built abdominal belt.

Ventral Hernia

The term *ventral hernia* is held to include all such ruptures of the abdominal parietes as do not appear at the inguinal, femoral, or umbilical apertures. Any portion of the wall may be the site of such a protrusion. For purposes of description the following varieties are recognised:—

1. Divarication of the recti.
2. Ventral hernia in the linea alba.
3. Ventral hernia in the linea semilunaris.
4. Ventral hernia following traumatism.
5. Lumbar hernia.

(1) *Divarication or Diastasis of the Recti.*—In children it is not infrequent to notice a widening and a thinning of the linea alba above the umbilicus. On crying or straining a semi-cylindrical protrusion occurs, and extends from the ensiform cartilage to the umbilicus. Clinically the condition is unimportant, and treatment is not necessary. In adults, and especially in multipara, the separation of the recti occurs below the umbilicus. The fingers can be readily passed into the gap. Rossetus relates a case occurring in a pregnant woman, in which the fœtus could be plainly felt in the hernial mass.

(2) *Ventral hernia in the Linea Alba* may occur above or below the umbilicus. The former is generally known as *epigastric* hernia, and occurs more frequently in men than in women. The linea alba above the umbilicus is about 3 mm. in width and consists of transverse fibres. It is no uncommon experience to find that these fibres are decidedly coarse in texture and small irregular apertures or spaces are left between them. Through these a protrusion of subperitoneal fat occurs, forming a rounded nodule readily felt on palpation. Such a mass may increase considerably in size and eventually drag outwards a peritoneal pouch, which may form a hernial sac, containing omentum, intestine, or both. Very rarely the stomach has been recognised as a hernial content.

These ruptures are chiefly remarkable for the persistent symptoms they induce. Dyspepsia, sickness, colic, and general intestinal discomfort are frequently complained of, and in not a few cases their cause may be overlooked or ignored. The treatment by operation should, if possible, be the routine procedure.

Below the umbilicus a ventral hernia, apart from those varieties to be mentioned, is very exceptional.

(3) *Ventral hernia in the Linea Semilunaris.*—A direct inguinal hernia is in reality a hernia at the linea spigelii. Apart from this form examples are rare. The majority of recorded cases have occurred below the umbilicus, and Mollière (whose name is attached to this rupture by French writers) suggested that the fold of Douglas determines the point of exit of the protrusion.

A very curious feature of herniæ in this situation has been described by Monro, Teale, and others. The hernial sac may enter the abdominal wall without passing through it, and consequently there is no manifest swelling on the body surface. The term "Masked Hernia" has been applied to this condition.

(4) *Ventral hernia following traumatism* may be found at any portion of the abdominal wall. Guthrie describes many cases occurring during the Peninsular War as the result of blows inflicted on the abdominal parietes. Any of the numerous operations practised upon the abdominal contents may leave a permanent weakness in the scar, which gradually yields under pressure from within. Cicatricial tissue is not suited to withstand pressure. Such herniæ can be almost entirely obviated by careful suturing, layer by layer, of the divided structures. Wherever possible, muscle-fibres should be separated in the direction of their length rather than cut. Mr. Howse first introduced this most important principle into surgical methods in performing gastrostomy, and MacBurney and others have carried out the same idea in operating upon the appendix. In every abdominal operation much can be done in this way to preserve intact the muscular structure.

The best treatment in the majority of cases consists in the use of appropriate specially-designed belts. An operation for the cure of such hernia is not attended with a large measure of success, and should only be employed in very favourable cases.

Lumbar Hernia

This form of hernia is rare, less than fifty examples being recorded. In more than half of these no mention is made of the precise point of exit.

In the lumbar region are two triangles, the triangle of Petit and the triangle of Grünfeldt, or the "upper" and "lower" lumbar triangles. The former is bounded by the anterior edge of the latissimus dorsi, the posterior edge of the external oblique, and, at the base, by the iliac crest. It is present in about 25 per cent of children and 75 per cent of adults. The latter is bound by the twelfth rib and the serratus posticus inferior above, the internal oblique below and in front, and the outer edge of the quadratus lumborum below and behind. The floor of the triangle of Petit is formed by a stout fascia covering the internal oblique, and beneath this by the aponeurosis of the transversalis. The upper triangle is roofed in by

the latissimus dorsi, and in the floor lies the lumbar aponeurosis alone; it is probably the weakest area in the lumbar region.

The triangle of Petit is the most usual point of exit of a rupture, being so mentioned in eleven cases; in three cases certainly, possibly in four, the hernia has passed through Grünfeldt's triangle. In the remaining cases the distribution has been quite irregular. As causes of the hernia may be mentioned traumatism, abscess formation, congenital defect of the muscles, and the presence of lipomatous masses derived from the subperitoneal tissue, which in their outward bulging drag small peritoneal pouches with them.

The hernia is rather more frequent in men than in women, and on the left side rather than on the right. The symptoms and signs are those of an ordinary hernia. The swelling is soft and globular and generally reducible. In a few cases strangulation has necessitated operation. It has been observed on dissection that the sac has returned with the contents when taxis was applied. The conditions for which hernia may be mistaken are abscess, hernia of muscle, sarcoma, lipoma, and hæmatoma.

Treatment can be carried out by means of an abdominal belt, by the wearing of a specially made truss, taking its fixed point from the opposite hip, or by operation. The latter has been practised by Owen, Coze, Giordano, and Zucker.

Obturator Hernia

An obturator hernia escapes from the pelvis at the opening for the obturator vessels and nerves. On the under surface of the horizontal ramus of the pubes is a groove which passes from behind, downwards, forwards and inwards, and according to Vinson has a length of 2 cm. Along this groove the bowel or other viscus passes, carrying before it the peritoneum, the subperitoneal tissue and the pelvic fascia. In its onward course the hernia may pass between the obturator membrane and the obturator externus, above the obturator externus, or beneath that upper slip which is occasionally separated from the body of the muscle by the passage of one or both divisions of the obturator nerve. The obturator nerve is generally found on the outer side of the sac and the artery behind, or behind and to the outer side. The most usual content of the sac is the small intestine; but the ovary, Fallopian tube, uterus, and bladder have all been found therein.

Symptoms and Signs.—The majority of the recorded cases have been recognised only after the onset of symptoms of acute intestinal obstruction. When a tumour is present it is most readily felt from the inner side of the thigh behind the adductor longus, when the limb is flexed, rotated outwards and adducted. A fulness in Scarpa's triangle may readily escape detection, especially in women, in whom this variety of hernia is very much more frequent than in men. In some cases the passage of intestine through the canal may be felt on rectal or vaginal examination. In one patient, examined by Macready, the finger, when made to sweep round the margin of the obturator foramen, was stopped by a firm cord, about the thickness of the thumb, which could be felt to enter the obturator canal. Pain may be elicited by making tense the obturator externus muscle. Disorders of sensation along the course of the obturator nerve are met with in about half the cases. There may be pain or numbness along the inner side of the thigh or in the groin, the hip, or the leg. This symptom of obturator neuralgia, generally called the "*Howship-Romberg*" *symptom*, even when present, has been frequently misunderstood, and may indeed be produced by other conditions than obturator hernia.

Treatment.—The mortality of strangulated obturator hernia is 85 per cent. If a hernia were diagnosed it would be almost impossible to adapt an effective truss. There is, therefore, every reason to urge that in all cases operative measures should be practised. The sac can be exposed by an incision parallel to the femoral vein and about 1 inch internal to it. The interval between the pectineus and the adductor longus is sought and the muscles separated or the former divided. Hæmorrhage, copious in quantity and difficult to control, has resulted from blindly incising the obturator membrane. In order to avoid this the structures should be exposed, and this procedure will be facilitated by the adoption of the Trendelenburg position and the abduction of the thigh on the affected side. In certain cases the reduction of the hernia through a median abdominal incision may be advisable.

Other varieties of hernia, such as ischiatic, pudic, pudendal, etc., are so rare that no description of them is required in this place.

Hernia of Special Viscera

There are some forms of hernia which derive their chief importance and certain of their intrinsic signs and symptoms from the inclusion of special viscera, such as the bladder, the ovary, and the vermiform appendix. These require, therefore, individual mention.

Hernia of the Bladder.—The frequency of bladder implication has been very differently estimated by various observers. As a rule it may be assumed that those operations wherein an opening up of the inguinal canal is practised are the most likely to reveal the bladder descent.

In 2543 operations for hernia tabulated by me in my "Arris and Gale" Lectures for 1900, there were twenty-three cases of bladder hernia,

giving approximately 1 per cent. Broadly speaking, it may be affirmed that this hernia is a disease of the aged and enfeebled. Only five cases are recorded as having occurred in children.

A hernia of the bladder may be present in the inguinal, femoral, perineal, obturator, and ventral forms of hernia. The inguinal is the most frequent, forming more than 90 per cent of the recorded examples.

Varieties of Cystocele.—Three varieties of cystocele are recognised :—

1. *Intra-peritoneal.*—An ordinary oblique inguinal hernia is present; into the sac a

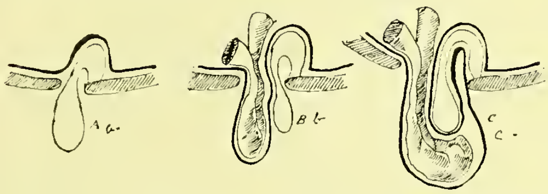


Fig. 11.—The three varieties of cystocele. A. Extra-peritoneal; B. Para-peritoneal; C. Intra-peritoneal.

portion of the bladder, *completely covered by peritoneum*, descends, the portion of the viscus implicated being the upper part of the posterior surface.

2. *Para-peritoneal.*—The inguinal hernia may be oblique or direct. On the inner side of the sac lies the bladder, in such manner that

Fig. 12.—Vesical hernia. Sir Astley Cooper's case. The bladder is covered by peritoneum except at the inner and posterior part. Para-peritoneal form. (Guy's Hospital Museum, No. 1170.)

the peritoneum of the inner wall of the sac is the serous covering of the outer wall of the bladder. The rest of the bladder outside the abdomen has no peritoneal coating. The viscus, therefore, is not a content of the sac, but projects into it. This form is the most frequent.

3. *Extra-peritoneal Cystocele.*—This form is rare. The bladder uncovered by peritoneum escapes from the abdomen on the inner side of the deep epigastric artery; the hernia, that is to say, is of the direct form.

The bladder may generally be recognised by the copious deposit of fat which surrounds it. In a very large number of cases the bladder has been wounded, being mistaken for the sac, a second sac, a cyst, or a lipoma.

Symptoms and Signs.—Very few cases of hernia of the bladder have been diagnosed before operation. When, however, a large portion of the bladder is implicated (the whole bladder, including the prostate, has been twice herniated) the symptoms are unmistakable. In such a case the signs are those of a hernia which at times contains fluid. The tumour is rounded, smooth, soft, fluctuating; the fluid is capable of reduction, and a desire to urinate is then created. The size of the hernia is subject both to rapid and to considerable alteration. The functional characteristics are striking. The chief of these is the "miction en deux temps," the divided micturition. A patient so affected voids urine and empties the portion of the bladder in the pelvis. Then, on pressure being applied to the hernia, the urine it contains is returned within the pelvic portion of the bladder and thence passes by the urethra. Micturition is in two stages and is associated with a simultaneous lessening of the hernial swelling. If the bladder, after being naturally emptied, is filled by the injection of lotion, the fluid will pass through the canal and distend the hernial tumour.

Treatment.—In operating upon all herniæ, especial attention should be paid to any thickening of the inner side of the neck of the sac, and more particularly to a deposit of fat in this situation, for by such signs is the presence of the bladder indicated. If the bladder be wounded, the wound should be stitched by a double layer of sutures and may then be safely reduced, and the operation completed in the usual manner.

As a truss cannot be safely worn in cases of cystocele, an operation should be resorted to whenever possible.

The accompanying table gives, at a glance, the chief features of the recorded examples of bladder hernia.

[Table

Hernia of the Bladder

Total recorded cases, 171

Cystocele diagnosed ; no operation . . . . 13 cases.
(In three of these, diagnosis was confirmed after death.)

Cystocele diagnosed ; operation . . . . 6 cases.
Bladder wounded (resection) 1
Bladder not wounded 5

Cystocele found ; after wrong diagnosis . . . . 4 cases.
(3 diagnosed as abscess ; 1 as sarcocele.)

Cystocele, first discovered at operation . . . . . . 107 cases.
Bladder recognised and not wounded 49
,, wounded and recognised 58
As follows :—
Wounded accidentally, though recognised . . . 2
Unexpectedly wounded 26
Intentionally incised 23
Being mistaken for—
The sac 3
A second sac 8
Tumour 4
Cyst 1
Omentum 2
Lipoma 2
Not mentioned . . . 3
(total 23)
Incised for diagnostic purposes 5
,, to confirm diagnosis 2
(total 58[1])

Cystocele, first recognised after operation because of wounding of bladder . . 22 cases.
Wound not noticed at operation 6
Supposed to be sac 9
,, ,, second sac 5
,, ,, lipoma 2

Cystocele, found post-mortem . . . . 19 cases.

[1] Bladder wounded and recognised 58

The cases were dealt with as follows :—

	No. of cases.	Result.		
		Healing without Fistula.	Fistula.	Death.
Bladder simply left open	2	0	2	0
,, stitched to skin	4	0	3	1
,, drained by tube	1	0	0	1
,, ligatured	3	0	1	2
Fastened in ordinary suprapubic incision, made for the purpose	1	0	0	1
Suture of Bladder—simple	10	5	2	3
,, ,, double layer	15	13	2	0
,, ,, triple layer	12	6	5	1
Suture ; no details	10	4	5	1
	58	28	20	10

Hernia of the Ovary.—Hernia of the ovary is very much more commonly inguinal than femoral, and congenital than acquired. Most of the cases are seen in infants, and the condition may then be bilateral, and be associated with malformations of the uterus, or the Fallopian tubes. In infants the condition may be only a temporary one, the ovary retiring spontaneously within the abdomen as the child grows. But at all ages irreducibility may be met with. According to Macready, 48·6 per cent are reducible in children and only 15·3 per cent in adults.

Symptoms and Signs.—In children the ovary may be felt in the inguinal canal or beyond the external abdominal ring as a small, oval, solid body, freely movable when pressed upon, and attached at its upper end by a thin cord which enters the abdomen.

In the adult the physical signs resemble those

given by an adherent pellet of omentum. Occasionally movement may be transmitted to the ovary by traction upon the uterus through the vagina. If the patient menstruates (which is not always the case), the ovary will be recognised as being swollen and tender at each period.

Examples of herniated cystic ovary and parovarian cyst have been recorded. (*See* "Ovaries.")

Treatment.—An operation is nearly always desirable. The ovary when healthy should be reduced if possible. If firmly adherent or altered by pathological processes removal will be necessary.

Congenital Hernia of the Cæcum and of the Sigmoid Flexure.—The cæcum on the right side and the sigmoid flexure on the left are occasionally present in "congenital" sacs. It has been shown that under such circumstances the visceral descent is the result of the action of the gubernaculum. Above the testis the smooth muscular fibres of the gubernaculum are continued upwards with the vessels of the testis and the vas deferens, in a peritoneal fold to which Lockwood has given the name "Plica vascularis." The ultimate attachment of these fibres is to the peritoneum lining the posterior abdominal wall. In these forms of hernia the unduly exaggerated action of the gubernaculum results in the dragging down bodily of the peritoneum to which the fibres are attached. There is, as it were, a sort of "landslip." The cæcum or the sigmoid flexure will then bear to the sac the same relation as to the parietal peritoneum before the descent began, for what was previously parietal peritoneum is now the sac, and the mutual relationship of viscus and serous membrane is unaltered.

Traces of the gubernaculum (the "natural fleshy adhesions" of Scarpa) have been discovered in the wall of the sac.

Treatment.—In the operative treatment of these cases the removal of the whole sac is, of course, out of the question. The peritoneum should be removed up to within an inch of the attachment of the viscus to the sac, the cut edges of the membrane stitched with a continuous suture of fine catgut, and the curtailed sac and its contents bodily returned.

Acquired Hernia of the Cæcum.—An acquired hernia of the cæcum is most frequently of the right inguinal variety, but right femoral, and left inguinal and femoral, are also met with. There has been much needless discussion as to the peritoneal coverings of the cæcum when involved in a hernia. It may be emphatically stated that there is no difference in the behaviour of the cæcum in this respect from that of any other viscus. The peritoneal covering of the cæcum and ascending colon varies within very wide limits. In some examples, though very few, the cæcum may have no coating of serous membrane on its posterior surface. In others, the cæcum and the whole of the ascending colon may be included within the layers of a mesentery continuous with that of the small intestine. Between these two extremes any condition may be met with, but in the great majority of cases, at least 95 per cent, the cæcum and a small portion of the ascending colon are completely clad with peritoneum.

Whatever the condition of the cæcum may be when within the abdomen, such will its condition be when descended into a hernia. If the serous covering be absent in the abdomen, it will be absent in the hernia; if complete within the abdomen, it will be complete within the hernia.

Hernia of the Appendix Vermiformis.—Hernia of the appendix derives its chief interest from the fact that inflammation, recurring from time to time, and possibly going on to suppuration or gangrene, may occur in the hernial sac. The implication of the little process in the hernia may lead to its strangulation or to an attack of appendicitis; or, it has been suggested, the occurrence of inflammation with the attendant swelling and stiffening of the process may be primarily responsible for the hernial descent.

The appendix may be alone in the sac, or be associated with the cæcum or with other viscera. It may be normal in appearance, and reducible; it may be bulky from the deposit of fat, distended into a cyst, or shrivelled and irreducible.

So far as the treatment of the condition is concerned, it is probably safer in all cases to remove the appendix. This can generally be effected through the incision made for the treatment of the hernia, but in certain cases a second incision over the cæcum may be desirable.

The Treatment of Hernia

The treatment of hernia is either *palliative* or *operative*.

Palliative Treatment consists in the wearing of mechanical appliances, specially fashioned for each separate form of rupture, known as *trusses*.

A truss should fulfil two essential conditions: it should retain the hernia completely under all circumstances, and it should be perfectly comfortable.

Inguinal and femoral trusses consist of a belt, containing a steel spring encircling the body and a pad, by means of which the force of the spring is applied to the point of exit of the rupture. Steel is better than any other metal, but is not wholly satisfactory. The secretion of the skin causes the spring to rust, and, after some time, to snap. No other material, however, is so satisfactory either in this or other respects. The pad is best made of cork covered with a layer or two of flannel. The truss throughout is covered on the external surface by leather or calf-skin, and on the inner side by chamois leather.

A patient, when measured for a truss, should be lying down. A tape is then carried round

the body from the base of the sacrum behind, between the crest of the ilium and the upper border of the great trochanter laterally, and just above the symphysis pubis in front. The number of inches, in this measurement, indicates the size of the truss.

A truss should be applied by the surgeon. The leaving of this matter in the hands of instrument-makers has led to much irregular practice, and to the introduction of many "special" or "patent" trusses which are, with scarcely one exception, worthless, or positively harmful. A truss is as surely a surgical instrument as a splint, and requires at least equal care and skill in the application.

INGUINAL TRUSSES. — 1. *For Bubonocele.* — A single truss should be of the exact size, obtained by measurement in the manner just mentioned; a double truss should be a size (that is, one

FIG. 13.—Inguinal truss to show the shape of the pad.

inch) larger, for its coverings are thicker and there is not the same adaptability as is afforded, in the single truss, by the strap. The inner end of the pad should be at the outer border of the rectus, and *above* (*not on*) the pubes. The truss should lie in the hollow just below the iliac crest, and should fit snugly and firmly. The commonest of all faults is to find a truss which is too large for the patient.

After placing the belt of the truss in position, the cross-strap should be first fixed, and then

FIG. 14.—Femoral truss to show the shape of the pad and the forward attachment of the under-strap.

the under-strap, which is carried round in the fold of the buttock from the shoulder of the truss to the bottom stud on the pad.

2. *For Scrotal Hernia.*—The ordinary pad is not sufficient, as a rule, to retain a rupture which has descended into the scrotum; it must be made fuller and more bulky, and of such a

FIG. 15.—Rat-tailed truss.

shape that the surface next the skin looks rather more in an upward direction. The soft part of the pad should also be prolonged downwards into a tail or under-strap which can be attached to a fixed buckle or hook in front of the shoulder of the truss. The double object of exerting pressure on the inguinal canal (by the pad) and on the external ring (by the under-strap) is effected by this truss, which is known as the rat-tailed truss.

3. *For direct hernia* a rat-tailed truss or a modification known as the "forked-tongue truss" is employed. In this latter the pad is carried inwards to the middle line and terminates in the strap which is fixed to the belt of the truss in front of the shoulder of the opposite side.

Double trusses should be made the same on both sides, double rat-tailed, double forked-tongue, or double ordinary, as the need may be. A truss whose two sides are dissimilar is rarely satisfactory.

FEMORAL TRUSSES.—The same measurement is used as in the case of inguinal hernia. The pad of a femoral truss is rather smaller than that of an inguinal, and is prolonged a little downwards to cover the femoral canal. The under-strap is attached to the truss well in front of the shoulder, so that when made tight it tilts the pad and causes increased pressure to be made on the femoral ring, which is normally almost horizontal when the body is erect.

The pad should be kept well outside the pubic spine and should not be allowed, as it often is, to over-ride that bony prominence. If the hernia is large and the pad has to be increased considerably in size in order to make effective pressure, a belt must be added to

encircle the thigh. If an inguinal hernia is present on the same side, the femoral pad can be prolonged upwards and inwards to cover the canal and ring.

Operative Treatment.—Since the introduction of modern methods into surgery the number of cases submitted to operation has, very properly, increased considerably. Probably, however, the question of the propriety of interference will remain for some time largely a matter of the personal choice of the surgeon. There are some surgeons who look upon most examples of hernia as legitimate cases for the "radical" cure, while others, carefully selecting their cases, have, by growth of experience, been led to believe that very few patients are really cured by operation, and they have in consequence restricted the treatment by operation to examples of congenital hernia. There are two points which should be considered before attempting any decision in the matter. The first is that, as shown by Macready and others, 36·1 per cent of cases of hernia, at first single, become, after the lapse of weeks, months, or years, bilateral. The second is that the recurrence of a hernia after radical cure is, unhappily, far more frequent than is generally conceded.

The results of Halsted and Bassini are the best so far recorded, but, allowing for the varying experience of all surgeons, one may state the likelihood of return as approximately 10 to 15 per cent of all the cases submitted to operation.

There are certain cases which, in the absence of urgent or striking reasons to the contrary, should be invariably submitted to the "radical cure." Such are—

1. Cases of uncontrollable or irreducible herniæ, where treatment by trusses is either inapplicable or has proved unavailing.
2. Cases occurring in young men, otherwise healthy and sound, desirous of entering the services.
3. Cases of strangulated hernia in which the gut has been reduced.

On the other hand, there are instances in which an operation is rigidly excluded. These are—

1. In the old, very feeble, cachectic or broken down, who are unable to tolerate any surgical treatment.
2. In young children, except in cases of strangulation.
3. In enormous herniæ, where a shrunken abdomen has resulted from the steady and progressive hernial enlargement.
4. In those cases where an inherited and extensive weakness of the abdominal muscles is the chief factor in determining the occurrence of the rupture.

Between these two groups, however, the majority of cases occur, and it is in dealing with them that the surgeon must exercise his discretion. My own feeling is that within reasonable age limits in healthy subjects, say from ten to fifty years, I should certainly advise treatment by operation in the very great majority of patients. If the patient is in good condition, if the rupture is of only moderate size, and if a complete union of the wound is obtained without suppuration, the chance of recurrence is very small indeed, probably less than 5 per cent. When, moreover, a recurrence under such circumstances does take place, the rupture is, almost without exception, easier to control by truss pressure than the ordinary complete hernia.

The Radical Cure of Hernia

1. *Inguinal.* — The treatment of inguinal hernia by operation was introduced by Celsus, who, it is interesting to note, expressly did not advocate any operative measures for ruptures which were strangulated. The removal of the testis, which for centuries was considered as a necessary incident in the operation, was advised by Paulus of Œgina.

Since the introduction of modern methods into surgical practice the operative treatment of all forms of hernia has been marked by a most florid activity. Several "essential principles" have been evolved, and the modifications of each have been bewildering in their frequency and unnecessary complexity. Probably no subject in the wide domain of medicine, not even the subject of pessaries, has had so much that is trivial and worthless written upon and around it as this.

Of the operations that have been widely used the following alone need mention :—

1. *Simple ligature of the neck of the sac* and removal (Socin).
2. Ligature of the neck of the sac and *stitching up the external ring* (Czerny, Banks, Championnière).
3. *Pleating of the sac* which is fixed as a pad at the upper end of the canal; suture of canal (Macewen).
4. *Torsion of the sac,* with suture in the canal (Ball).
5. Torsion of the sac, *displacement of the neck,* suture of canal (Kocher). Recently modified (see description later).
6. Removal of the whole sac, *displacement of the cord,* suture of the canal (Halsted, Bassini).

There can be no question that the operation most widely practised at the present time, and incomparably the most successful in results, is that introduced by Bassini. The operation of Halsted (which has priority of publication) is the same in principle, but the details of the operation are different in certain particulars.

Bassini's Operation.—An incision is made in the inguinal region, beginning a little below

and internal to the anterior superior spine, and ending at the centre of the external abdominal ring. The incision exposes the tendon of the external oblique muscle, which is split, in the direction of its fibres, from the ring upwards for about $1\frac{1}{2}$ inch.

The sac is now sought for among the structures of the cord. If the hernia has never presented at the external ring this may be a matter attended with some difficulty, but a little patient clearing of the cord will usually suffice to expose the sac, which is then gently and carefully isolated up to the internal abdominal ring. The sac is now opened, the intestine or other viscus replaced within the abdomen, and the omentum, if present, removed. The sac is tied at the internal ring after transfixion and removed; or the sac is removed, and the cut edges of the parietal peritoneum at the neck stitched by interrupted or continuous sutures. The cord is then gently lifted from its bed, and held up by blunt hooks. Sutures, three, four, or five in number, are introduced through Poupart's ligament on the outer side, and on the inner through the arching fibres of the internal oblique and transversalis, the transversalis fascia, the conjoined tendon, and, if necessary, the sheath of the rectus also. The sutures are passed underneath the cord; when tied and cut short the cord is laid upon them. Finally the cut edges of the aponeurosis of the external oblique are united over the cord by a continuous suture, and the skin wound is closed. A drainage tube is not necessary. If, on laying open the canal, the veins of the cord are found to be varicose, they should be excised. At times the fat lying along the cord, continuous above with the subperitoneal fat, is greatly increased in bulk, forming a definite lipoma which must be removed. Under all circumstances the cord should be handled with care and gentleness; a rough and hasty stripping and tearing of the sac may lead to inflammatory trouble in the wound or in the testis.

Fig. 16.—Transplantation of the rectus muscle. (Bloodgood.)

If the cord is found to be so thick that the aperture left for its passage through the abdominal wall might tend to induce a fresh descent of the hernia, the cord may be split into two equal portions, the outer one being placed outside the upper stitch, and the inner between that stitch and the next. If the cord is turgid with veins which are not varicose this method of splitting is preferable to the excision of the veins, for experience has abundantly shown that the latter procedure is one not seldom attended by an attack of acute or subacute orchitis, which may, itself, be followed by a gradual withering of the testis.

When a recurrence of the hernia takes place after this operation it is generally found at one of two points, at the internal abdominal ring or at the inner and lower portion of the external abdominal ring. When at the former point it is most probably due to the bulky thickening of the cord, and may be prevented by the splitting of that structure in the manner just described. When at the latter point it is almost certainly due to the weakness of the conjoined tendon, a weakness which, as I have already mentioned, is very frequent. In order to strengthen the abdominal wall at this point the following device is useful. Before introducing any of the sutures, the sheath of the rectus muscle is opened by an incision along the outer side in the direction of the length of the fibres. The muscular bundles of the rectus are then included in the two or three innermost stitches. On tightening these stitches the muscular fibres are brought into close approximation with Poupart's ligament, and the inner end of the gap in the abdominal wall is thereby considerably strengthened. This so-called "transplantation of the rectus" was first suggested by Wöfler in 1892, but his paper has not received general recognition. Though I have practised this method for over two years I was myself unaware of Wöfler's article until I saw it quoted in Bloodgood's recent work.

Halsted's Operation. — After exposing the arching fibres of the internal oblique, as in Bassini's operation, the free lower border of this muscle is caught with two artery clamps, placed 1 cm. apart, and the muscle cut between them for a distance of 3-4 cm., at right angles to the muscular bundles. The division of the muscle is made as far from the linea semilunaris as possible. The cord is brought out through the incision in the internal oblique and is made subcutaneous. Five Mattrass stitches, as a rule, are introduced, passing through the aponeurosis of the external oblique, the internal oblique, transversalis muscle and fascia on the one side, and through the transversalis fascia, Poupart's

ligament, and fibres of the aponeurosis of the external oblique on the other.

Kocher's Operation.—Dr. Albert Kocher informs me that the operation now practised (May 1900) by Prof. Kocher is carried out as follows :—An incision is made over the inguinal canal ; the sac is isolated from the cord very carefully as high up as possible, traction being maintained upon the sac in order to pull it down to its farthest extent. A special light forceps then seizes the end of the sac, which is invaginated upon itself (just as one would turn the finger of a glove inside out), and brought up, about 1 cm. above and outside the internal abdominal ring. An incision of $\frac{1}{2}$ cm. is made through the external oblique aponeurosis down to the point of the forceps, which is thrust through the small wound. The sac, still inverted, is pulled out as far as possible. A silk ligature is passed round the neck of the sac and the sac removed beyond the ligature. The stump of the sac is pushed back through the opening in the fascia. The ligature surrounding the neck of the sac is left long, and with it the edges of the little wound in the aponeurosis (including the cut edges of the parietal peritoneum there) are stitched together.

Finally some six or eight sutures are passed through the anterior wall of the canal to strengthen it, and so make the outer ring smaller.

For the buried sutures many materials have been recommended. Halsted uses silver-wire and is eminently satisfied with it. Bassini, since 1892, has abandoned the use of silk, and now employs chromicised catgut. Silk, silkworm gut, kangaroo tendon and chromicised catgut are those in general use. Silk has the great disadvantage that, if the wound becomes septic, the silk will be readily infected, and thereby give rise to a persisting sinus. Silkworm gut is open to the same objection, but in practice I have had very little trouble with it. Kangaroo tendon and chromicised catgut both remain unabsorbed for several weeks, and probably in that time the deep union of the wound is sound and complete. Marcy and Bull and Coley use and advise kangaroo tendon. After trying impartially all these forms of suture I now use nothing but silkworm gut.

In all operations for the radical cure of hernia a point of chiefest importance is the securing of perfect primary union. In no other operation is this so essential. Recurrence of the hernia is proportionately very much more frequent in cases that have suppurated than in those where primary union has been secured.

2. *Femoral.*—The radical cure of a femoral hernia is generally very much more satisfactory in its result than is the case with any other variety of rupture.

The operative procedure is very similar to that employed in cases of inguinal hernia, so far as the earlier steps of the operation are concerned. A vertical or transverse incision is made, the sac exposed, stripped and opened. The viscera are replaced or omentum removed,

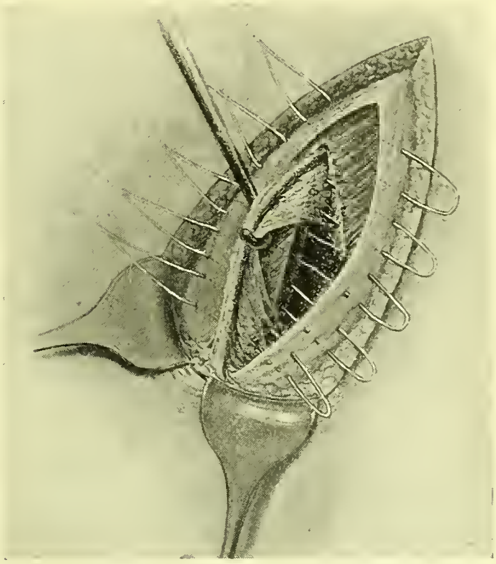


Fig. 17.—*Halsted's operation.* Showing the division of the internal oblique, the transplantation of the cord, and the method of introducing the stitches. (Bloodgood.)

the sac ligatured as high as possible and removed. For the closure of the canal and ring two methods are in general use :—

A. *Bassini's Method.*—With a curved needle a series of sutures are introduced uniting the fascia covering the pectineus muscle with the inner end of Poupart's ligament, above and internally, and with the falciform process of Burns externally. All the sutures (five or six are generally used) are passed and are then tied from above downwards.

B. *Lockwood's Method.*—A special curved needle armed with silk is used, whose point is guided up the femoral canal until it rests against the inside of the linea ilio-pectinea, opposite the outer edge of Gimbernat's ligament. The needle is then rotated so that its point scrapes over the linea ilio-pectinea and picks up Cooper's ligament. Finally the point emerges through the upper part of the pectineal fascia, where it is unthreaded and withdrawn, leaving the suture beneath Cooper's ligament. Additional sutures are passed in exactly the same way, but each a little farther outwards, until the last lies at the inner edge of the common femoral vein. Two or three sutures generally suffice, but more may be necessary. The next step is to again thread the upper end of each ligature in turn through the herniotomy needle, and by pushing the point of the needle half-way up the femoral canal and rotating it forwards pass the thread from within outwards through Hey's ligament, close to its junction with Poupart's ligament. On tying these

sutures Hey's ligament is united to Cooper's ligament, and the crural canal is firmly closed.

Lockwood's operation is most satisfactory in its results, and is, in my opinion, decidedly better than Bassini's, which closes only the lower opening of the canal, and leaves untouched the crural ring.

Herniotomy. *See* Hernia (*Treatment, Operative*).

Heroin.—Diacetyl-acid-ester of morphine; a white crystalline powder with a slightly bitter taste. It is insoluble, and is usually employed in the form of *heroin hydrochloride*, which is freely soluble. *Dose*—$\frac{1}{20}$-$\frac{1}{6}$ gr. It is somewhat similar in its action to codeine. It does not stupefy the patient, does not constipate, and is stimulant rather than depressant to respiration. It is given to check excessive coughing in phthisis, bronchitis, and asthma. *Glyco-Heroin*, a proprietary article, adult *dose* ʒi., is a convenient preparation for such conditions. Heroin is preferable to morphia for continuous use in inoperable malignant disease, etc., when it may be administered by the mouth, hypodermically, or as a suppository according to the indications present.

Herpes.

See also Chest-Wall, Affections of (*Pain, Herpes Zoster*); Cornea (*Phlyctenular Ulcer, Herpes of the Cornea*); Dermatitis Herpetiformis (*Herpes Gestationis, etc.*); Erythema (*Types, Herpes Iris*); Eyelids, Affections of (*Herpes Frontalis*); Foot and Mouth Disease (*Diagnosis, Herpes Labialis*); Gout (*Irregular, Cutaneous System*); Hysteria (*Disorders of Circulation and Trophic Disorders*); Malaria (*Benign Tertian Fever, Febrile Paroxysm*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms, Skin Lesions*); Nerves, Peripheral (*Injuries, Symptoms*); Nerves, Multiple Peripheral Neuritis (*Symptomatology*); Penis, Surgical Affections of (*Inflammatory, Herpes Progenitalis*); Pharynx, Acute Pharyngitis; Pneumonia, Clinical (*Clinical Features, Herpes Labialis*); Pregnancy, Affections and Complications (*Cutaneous Rashes, Herpes Gestationis*); Puerperium, Pathology (*Affections of Nipples, Herpes Zoster*); Purpura (*Purpura as a Disease, Symptoms*); Skin Diseases of the Tropics (*Dhobie Itch or Herpes Circinatus; Tinea Imbricata or Herpes Desquamans*); Stomatitis (*Aphthous*); Tabes Dorsalis (*Symptomatology, Sensory Symptoms*); Venereal Disease (*Soft Sores, Diagnosis, Herpes Progenitalis*).

Herpes

Herpes is a term employed to designate an acute inflammatory affection, characterised by the development of vesicles closely grouped upon a patch of erythematous skin or mucous membrane.

Two forms of disease are recognised as coming under the above anatomical designation, but they differ widely as regards their clinical phenomena and pathological relations.

Herpes Febrilis vel Catarrhalis

Febrile or catarrhal herpes frequently follows a rigor, and may be its sole sequela. Before the appearance of the eruption the temperature may be considerably raised, especially in children, but it usually falls on its appearance. Crops of herpes are of common occurrence in acute febrile diseases, accompanying their onset or crisis; they are most frequent in pneumonia, pleurisy, ague, enteric fever, and "catheter fever," but may accompany many indefinite gastro-intestinal disturbances. In other instances herpes may occur and recur frequently, often at definite intervals, without any rise of temperature or tangible determining cause. Many cases tend to occur in spring and autumn, and attain almost "epidemic" proportions. The lesions of febrile herpes do not show nerve distribution like those of zoster, from which they further differ in being frequently bilateral, sometimes even symmetrical, in being painless, leaving no scar, and in their great tendency to relapse.

Herpes facialis is the commonest form; it attacks in order of freqnency the lips, the alæ nasi, the chin, and the ears; the "flush patches" in the malar region are also often involved. Patches may also appear on the tongue, soft palate, uvula, and tonsils generally in connection with digestive disorders, and as these soon give rise to minute ulcerations ("dyspeptic ulcers"), as the result of the maceration of their epidermis by the fluids of the mouth, they may be mistaken for aphthæ, diphtheria, or even syphilis. On the skin a slightly infiltrated reddish patch is at first present, accompanied generally by a little tension, burning, tingling, or itching. The patch is usually round or oval, and may be of very variable size; in a few hours minute vesicles form upon it, averaging about the size of a millet seed. Some of these vesicles usually coalesce, but at the margin they generally remain discrete. In two or three days the vesicles burst, their epidermis shrivels, their liquid contents dry up to form a thin scab; when this is removed a minute, shallow, poly-

cyclical ulcer is exposed. At the end of a week or so the scab, which becomes daily thinner, finally separates, leaving only a pinkish or slightly pigmented spot which rapidly disappears. It is extremely rare, and in marked contrast to zoster, to have any permanent scar left even when the lesions have been the seat of pus infection, or been irritated by unsuitable dressings.

Herpes genitalis is a common disease in the male sex up to forty years of age, but in the female sex its incidence is almost entirely confined to prostitutes, occurring often at their menstrual periods, and frequently in those who have suffered from antecedent syphilis. In the male the disease usually occurs on the prepuce; it tends to relapse at much shorter intervals than herpes facialis, and is certainly of frequent occurrence in persons who have had syphilis. It sometimes recurs after every act of coitus in some individuals, irrespective of the health of the woman, and often follows indiscretions of diet, or may occur independently of any ascertainable cause. In France it is regarded as strong evidence of "arthritism" (*vide* "Gout," vol. iii. p. 494). The chief importance of progenital herpes lies in the facility for which it may be mistaken for soft chancres, especially when not seen at the commencement. Generally speaking, the lesion of chancroid is at first single, those of herpes are multiple, and on an erythematous base; the herpetic lesions are generally cleaner, not so purulent as the chancre; there is more burning and itching in herpes than in chancroid; finally, the pus of herpes is not auto-inoculable.

Treatment.—Facial herpes may often be abated in the early stage by mopping with swabs soaked in rectified spirit or eau de Cologne. Sometimes bathing with very hot water answers equally well, or the application of a coating of collodion.

In the later stages inert powders (oxide of zinc, starch, chalk, emol) may be dusted on; when the vesicles burst, dilute boric vaselin or ammoniated mercury ointment may be used. Norman Walker recommends painting the affected parts in each attack of facial herpes with argent. nitrat. (gr. xx.) in spir. æther. nitrosi (℥j.), as distinctly increasing the intervals between the attacks and ultimately sometimes effecting a cure. For the same purpose arsenic in prolonged courses and gradually increasing doses is warmly recommended in many quarters, while others rely upon nerve tonics (quinine, strychnine, belladonna, bromide of potassium) or change of air to various spas, home or foreign. In the management of genital herpes in both sexes strict attention to cleanliness is of paramount importance.

Herpes Zoster or Zona

Herpes zoster, zona, or shingles—so called from its girdle-like distribution—is a fairly common disease, and occurs in about 6 per cent of all skin cases (Radcliffe-Crocker).

Its etiology is obscure, and beyond cold or chill no definite cause can be assigned for it, although recent views tend to favour the possibility of its being caused by a specific poison. It occurs in both sexes, and is generally stated to be most frequent between the ages of 3 and 20, the maximum incidence being between the ages of 12 and 13; but older people are by no means exempt, and in them the symptoms are apt to be severe. Although the disease often occurs in persons in robust health, general debility acts as a predisposing cause, as also—as first pointed out by Hutchinson—does arsenic when taken over long periods of time; but that arsenic is only an indirect cause is clearly shown by the small number who are affected out of the multitude who take the drug.

Herpes zoster may be *symptomatic*, and, as such, is seen in certain diseases of the nervous system, as tabes dorsalis and myelitis; in the latter the eruption often corresponds to the upper level of the anæsthesia. Chronic peripheral irritation, *e.g.* caries of a rib or nerve injuries, will also sometimes give rise to attacks, which in such cases may be recurrent. The disease is probably of a specific nature, as shown by its acute onset, definite course, the infrequency of second attacks, and by the fact that it often recurs in distinct epidemics. Head considers it an acute inflammatory affection of the posterior root ganglia, analogous to that of the cells of the anterior cornua in acute anterior poliomyelitis.

Symptoms and Course.—An attack of herpes zoster is usually ushered in with general feelings of malaise and a rise of temperature which, in children, may reach 102° F.; superadded to these symptoms being acute pain and hyperæsthesia over the nerve area subsequently to be affected. In children the amount of pain may be slight or nil. The date of the appearance of the eruption is variable, but it usually appears at earliest two or three days after the first symptoms have shown themselves.

The eruption appears first as an erythematous patch or patches, upon which numbers of small vesicles develop within a few hours. This outbreak follows the distribution of the nerve-fibres of the posterior roots, and is most intense where the main branches come to the surface, the patches being frequently oval, their long axis parallel to the subjacent nerve. The characteristic distribution is most typically shown in a case of intercostal herpes, in which one group of vesicles will often be seen near the spine over the spot where the posterior primary branch of the posterior root comes to the surface; another close to the axillary line over the site of the lateral branch of the anterior

primary division; and a third group near the mid-line marking the termination of the anterior primary branch. There is no uniformity as to the order in which these maxima of intensity are affected, but as a rule the first patch to evolve is that nearest the nerve centre. The eruption is always most intense at these spots, and the intervals between them may or may not be filled up according to the severity of the attack. The right side is, curiously enough, much more commonly affected than the left. The corresponding lymphatic glands are almost always enlarged and tender, and may be so affected before the appearance of the eruption.

The eruption may continue to appear in crops extending over a week, or even longer, or it may be fully developed in two days; in the former case it appears early in the disease, and its evolution is accompanied by persistent pyrexia and malaise: in the latter it usually denotes the termination of the pyrexial period, and may be regarded as a "critical" phenomenon.

The initial lesions are erythematous patches, upon which papules and small vesicles rapidly form, which by coalescence may form comparatively large blebs. Some difficulty in diagnosis may occasionally be caused by the eruption being abortive, and appearing only as ill-developed grouped papulo-vesicles.

The vesicles are, at first, filled with clear fluid, which gradually becomes turbid, and dries up to form small scabs, which usually separate in about ten days. In more severe cases the vesicles, probably from extraneous causes, contain purulent or blood-stained fluid, often become gangrenous, and give rise to ulcers of various depths, which only heal with considerable scarring.

Herpes zoster may occur in the course of any cutaneous nerve, and is designated by various names according to the part affected, *e.g.* herpes ophthalmicus, cruralis, etc. It is especially frequent over the distribution of the intercostal, lumbar, and ophthalmic divisions of the fifth nerve, and is very rarely found below the knee. Herpes is comparatively frequent over the areas of distribution of the sciatic, anterior crural, ilio-inguinal, musculo-spiral, occipital, and superficial cervical nerves. Herpes of the ophthalmic division of the fifth nerve is especially important, as the subsequent scarring is generally deep and correspondingly disfiguring, and as the cornea is apt to be attacked, a complication which is most frequent in cases where the nasal branches of the nerve are affected. Zoster of the superior and inferior maxillary nerves is generally accompanied by lesions on the soft and hard palate, and on the tongue.

There are several very striking and important characteristics of attacks of zoster: the eruption is very seldom bilateral—though it may occasionally overlap the middle line—and it is never symmetrical; one attack almost invariably protects against a second, and in those cases in which second attacks do occur the eruption never occurs over the site of the former lesion.

Distribution.—Herpes zoster follows the distribution of the fibres of the posterior root ganglia, and not the course of peripheral nerves. This important fact was first pointed out by Baerensprung, and has recently been further extensively investigated by Head, who has used the distribution of the eruption as a means of mapping out the areas of skin supplied by the different posterior nerve roots.

Pathology.—Each vesicle consists of a small unilocular cavity containing fluid and altered epithelial cells with leucocytes; the papillæ of the skin form its floor. Several vesicles may coalesce to form multilocular blebs. Protozoa-like bodies described by Pfeiffer as pathogenic are probably only altered epithelial cells. The underlying papillæ are deeply congested and infiltrated with leucocytes. The peripheral cutaneous nerves show swelling of the neurilemma, degeneration of the medulla and moniliform swelling of the axis-cylinder. Ten days after the onset of eruption the larger branches show marked degenerative changes (Campbell and Head). Changes have now been definitely established occurring in the ganglia of the posterior nerve roots which extend for some distance up the posterior columns of the cord; the most common conditions found are hæmorrhages and inflammatory exudation, and secondary to these sclerosis, while degenerative changes have been traced from the posterior root ganglia through the posterior roots to the peripheral sensory nerves. In herpes ophthalmicus inflammatory changes are present in the Gasserian ganglion.

Complications and Sequelæ.—The amount of *scarring* varies considerably; in some cases there is scarcely any, and in others it is very deep, the preceding ulceration being sometimes very persistent.

Pain in the majority of cases disappears with the rash; in some, it persists for a considerable time after, and occasionally lasts for months or even years. This after-pain is a very troublesome symptom, which is of very common occurrence in elderly people, and in those cases where the disease has been followed by deep scarring.

The *ocular complications* of herpes of the ophthalmic division of the fifth are not infrequently serious, the most common being ulceration of the conjunctiva and cornea; iritis and panophthalmitis occasionally occur, and in very rare instances paralysis of orbital or ocular muscles have been met with.

Other motor paralyses are occasionally observed. Thus paralysis of the hand has been

recorded after a herpes involving the cervical and post-dorsal nerves; facial paralysis of Bell's type has been stated to follow cervical herpes. On the other hand, Head states that "with the exception of the iris, there is no evidence that any deep structure (pleura, peritoneum) is affected during an attack of herpes zoster."

Occasionally the sensation is impaired for a time over the affected area of skin, but it does not usually persist; and Head found that the impairment was generally most marked over the areas of deepest scarring; thermal sensations according to Head suffer more than others. The writer has observed several well-marked instances of "anæsthesia dolorosa" as a particularly obstinate sequela.

Diagnosis.—The diagnosis does not usually present any difficulty, but before the appearance of the eruption a pleurisy or pleurodynia may be closely simulated or in cases of herpes ophthalmicus a *tic douloureux*. The collections of grouped vesicles on erythematous bases with their definite lines of unilateral distribution usually form a characteristic picture; but sometimes doubts may arise from the lesions being abortive. Again, if the condition is seen for the first time when the vesicles have lost their early characters and run together into indefinite areas, the condition may resemble eczema, and the primary lesion may be further masked by secondary infections. Occasionally when the disease has occupied unusual regions, the erythema and vesicles have been mistaken for erysipelas.

Cases described as universal herpes zoster are undoubtedly instances of dermatitis herpetiformis (*q.v.*). In all cases of doubt a careful inquiry into the history of the onset and the accompanying symptoms will usually clear the matter up.

Treatment.—There is no marked method of treatment known which will certainly cut short the disease. The indications are to treat the patient on the same lines as for any other acute febrile affection, and to protect the affected parts from the air and from rupture. For this purpose the eruption may be freely dusted with zinc oxide and starch powder, and covered with a thick layer of medicated cotton wool and a bandage. The application of Unna's glyco-gelatin of zinc is often very efficacious, apparently arresting the eruption. When the pain is severe a small quantity of powdered opium added to the dusting powder will sometimes give relief. The application of camphor-chloral or belladonna liniment may also be useful.

Generally speaking, the use of ointments or greasy substances is unsuitable, but if the vesicles burst, a boric ointment containing cocaine is valuable.

As soon as the attack is over, the patient should be given general tonics, such as strychnine, iron, quinine, or arsenic, with a generous diet and sound wine; while cod-liver oil is often of signal service. When the pain persists it becomes a very troublesome symptom; antipyrin, phenacetin, and other drugs of this class may be given, and the continuous current is frequently efficacious, but too often morphia is the only drug which gives any real or permanent relief.

Herring. *See* Snake-Bites and Poisonous Fishes (*Clupea Thryssa, etc.*); Toxicology (*Fish, Herrings*).

Hesperidene.—A glucoside ($C_{18}H_{21}O_9$), obtained from bitter orange rind; a variety of limonene.

Hesselbach's Hernia.—A rare variety of femoral hernia in which a series of diverticula is sent off from the sac through the openings in the cribriform fascia. *See* Hernia (*Femoral, Varieties*).

Hesselbach's Triangle.—The triangular interval formed by the plica epigastrica, the outer edge of the rectus abdominis, and Poupart's ligament. *See* Hernia (*Inguinal, Anatomy*).

Heteradelphus.—A variety of double monster, in which one fœtus is fully formed, while the other is little more than a parasite attached to the former (*e.g.* an acephalous mass attached to the thorax of the autosite).

Hetero-.—In compound words *hetero-* (Gr. ἕτερος, the other, or one of two), means "different," and is used in opposition to *homo-*, or *auto-*, or *iso-*, or *ortho-*; thus *heteroblastic* means arising from cells of a different kind, in opposition to *homo*blastic, arising from cells of the same kind.

Heterocele.—A hernia contained in a prolapsed part of such an organ as the rectum.

Heterocephalus.—A double monster in which there are two heads, one of which is incompletely developed.

Heterochromia.—This name is usually given to a pigmentary anomaly of the iris: that of one eye may be different in colour from that of the other eye, or one part of the iris may be differently coloured from another. *See* Iris and Ciliary Bodies (*Congenital Abnormalities of the Iris*).

Heterochronism.—A term applied specially to the appearance of certain symptoms in a malady at a time other than that at which they usually manifest themselves, or to the development of organs or tissues at abnormal times; "displacements in time."

Heterochthonous.—Arising outside the organism, in opposition to *autochthonous*.

Heterocinesia. *See* Hysteria (*Motor Disorders*).

Heterodymus. — An asymmetrical double monster in which the parasitic fœtus (consisting of little more than a head) is attached to the thorax of the autosite.

Heterogeneous.—Of a mixed character, in opposition to *homogeneous*; thus a heterogeneous tumour is one consisting of tissues derived from the different layers of the blastoderm.

Heterologous.—Usually employed in the sense of being different from the normal tissue of the part.

Heteromorphous.—Of abnormal form or structure; of the nature of a malformation or anomaly.

Heteropagus. — An asymmetrical double monster in which the imperfect parasitic fœtus (with a rudimentary head) is attached to the thorax of the autosite.

Heterophonia. — An abnormality in voice, or the articulation of words different from those intended to be used (*heterolalia*).

Heterophoria. *See* Strabismus (*Concomitant Squint*).

Heteroplasty.—A surgical procedure in which portions of one organism are transplanted into another, *e.g.* a piece of ovary from one woman to another ("heteroplastic ovarian grafting"), or a piece of skin from one person to another as in the Indian form of rhinoplasty.

Heteroproteose. *See* Physiology, Food and Digestion (*Gastric Digestion, Proteolytic Period*).

Heteroscope.—A device for the orthoptic treatment of strabismus. *See* Strabismus (*Treatment, orthoptic*).

Heterotaxy.—Inversion or transposition of the viscera; situs inversus; situs mutatus. *See* Teratology (*Heterotaxy*).

Heterotopia.—Displacement of an organ or tissue (*e.g.* the occurrence of the grey matter of the spinal cord between the white columns).

Hetero-typus. — An asymmetrical double monster in which the parasitic fœtus (more or less defective) is attached to the anterior aspect of the autosite.

Hetol.—The cinnamate of sodium, recommended (in the form of intravenous or of local injections) in tuberculosis, alone or in association with *hetokresol* (cinnamic-acid-metakresyl-ester).

Hetralin. — Dioxybenzol hexamethylenetetramine; recommended as a urinary antiseptic, in daily doses of from 24 to 45 grains, in gonorrhœa, cystitis, etc.

Hexadactylism. — Polydactylism in which the number of digits is increased to six.

Hey's Amputation. *See* Amputations (*Lower Extremity*).

Hiatus.—An opening or cleft, *e.g. hiatus diaphragmatis aorticus*, the foramen in the diaphragm through which the aorta passes.

Hiccough.

See also Alcoholism (*Visceral Variations*); Brain, Tumours of (*Symptoms, Respiration*); Cholera, Epidemic (*Symptoms, Algid State*); Diaphragm (*Spasm*); Gastro-Intestinal Disorders of Infancy (*Disorders of Digestion, Hiccough*); Hypnotism (*Therapeutic Uses*); Liver (*Perihepatitis, Symptoms*); Mediastinum (*Growths, Symptoms*); Physiology, Respiration (*Special Respiratory Movements*); Spasm (*Hysterical*); Spine, Surgical Affections of (*Fracture-Dislocation, Symptoms*); Typhoid Fever (*Complications*); Uræmia (*Symptoms, Respiratory*).

Hiccough; Hiccup; Singultus. Hiccough is usually described as being produced by a sudden descent of the diaphragm, accompanied by a spasmodic closure of the glottis, the characteristic sound being due to the inrushing column of air striking against the closed vocal cords.

While there is no doubt as to the part that the diaphragm plays, the exact condition of the glottis is more open to question. Poore considers that the assumption of a spasmodic closure of the glottis is not warranted, and it certainly seems difficult to understand why any such closure should occur, when it is remembered that during the descent of the diaphragm in ordinary respiration the glottis is dilated. It seems reasonable to suppose, therefore, that the closure during hiccup is only a *relative* one, due not to any spasm of the adductors of the larynx, but rather to a want of action of the dilators, the correlation between these latter and the diaphragm ceasing to exist when the diaphragm contracts irregularly and serves no useful part in respiration.

Etiology.—Hiccough may be central or peripheral in origin. Irritation of the phrenic nerve in any part of its course may give rise to irregular diaphragmatic contractions, and in this way hiccough has been produced by tumours and other irritating lesions of the neck.

Most commonly, however, hiccough is a reflex act, the pneumogastric usually being the afferent nerve. Flatulence, overloading of the stomach,

excess of alcohol, and the different forms of dyspepsia, are among the more common causes. It is also frequently associated with peritonitis, cardiac failure, uræmia, and other acute diseases, as typhoid fever, pneumonia, and pleurisy. Richardson has suggested that its presence in acute diseases is frequently due to the alcohol which is prescribed. Tobacco smoking occasionally appears to be a cause. In an obstinate case recorded by Stevenson large caseous masses were found after death at the root of each lung, which it was thought might have involved the phrenic or vagus nerves. In chronic cases recurrences are sometimes prone to take place at the menstrual periods.

Hiccough occasionally occurs as a true neurosis independently of any definite cause. In a case recorded by Ottoni, a patient, after suffering for some years from hysteria, suddenly became subject to the most obstinate hiccup, which, at the time the case was reported, had existed for thirty-four months, with rare intervals of ease, and it was always found that any temporary amendment was followed by redoubled severity.

Liveing mentions instances where the hiccup appeared to bear some relation to epileptic fits, attacks apparently occurring in place of a fit.

Symptoms.—The symptoms of hiccup are too well known to need any detailed description: the sudden inspiration, accompanied by the characteristic sound, is familiar to every one. Usually an attack causes only a slight temporary inconvenience, and either passes off spontaneously or is cured by some simple remedy. Occasionally, however, the condition is most obstinate, and the exhaustion produced by the constant repetition of the muscular efforts and want of rest becomes very serious, especially if the patient has already been weakened by existing disease. The length of time the symptoms last, and their severity, are very various. A case recorded by Foot gives a good idea of the severity of the symptoms which can occur. The patient, a boy aged 15, hiccoughed incessantly, except when he was asleep, for twenty-six weeks. The onset of the attack was quite sudden, and he had previously had two other but much less severe attacks. The rate of hiccough was calculated on thirteen different occasions in this case. It varied from 8 to 22 per minute, and from 480 to 1320 per hour, the average being 14 per minute, or 840 per hour.

The significance of hiccough varies with its cause. Most commonly it signifies a slight digestive disturbance, and is of no importance. When it occurs during acute diseases it is usually a grave omen, especially in cases of peritonitis.

Treatment.—Ordinary attacks of hiccough either soon pass off spontaneously, or are readily amenable to some of the familiar remedies, such as holding the breath, sipping a glass of water, or taking several deep respirations at regular intervals. In cases which do not readily pass off, a draught containing aromatic spirits of ammonia or warm applications to the abdomen may be tried, or, if the stomach is obviously overloaded, an emetic may give the desired relief.

The more obstinate cases, unfortunately, often fail to yield to any of these remedies, and it would appear from records that no one drug can be relied upon in any given case. Among the preparations which seem to have been most successful in curing the complaint, nitro-glycerine, ether, turpentine, and liquid extract of ergot may be mentioned. Most observers are agreed that morphia exercises but little influence in checking the condition. Foot's case, which has already been referred to, recovered on pills of iodoform, extr. of Indian hemp, and extr. of conium. Under this treatment the hiccough diminished and finally ceased. Mechanical measures, such as blisters over the origin of the phrenic nerve and pressure upon its trunk at the root of the neck, and depression and traction of the tongue, have been practised, and the last-named method appears to have been successful in several cases which had failed to yield to medicinal treatment.

Hidroadenitis. *See* HYDROADENITIS.

Hidrocystoma. *See* HYDROCYSTOMA.

High-Frequency Currents. *See* X-RAYS AND HIGH-FREQUENCY CURRENTS.

Highmore, Antrum of. *See* NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Anatomy, Maxillary Sinus*).

Highmori, Corpus.—The incomplete septum formed in the testicle by prolongations from the tunica albuginea into its substance. *See* PHYSIOLOGY, REPRODUCTION (*Testis, Supporting Framework*).

High-Tension Pulse. *See* PHYSIOLOGY, CIRCULATION (*Arterial Pulse, Tension*); PULSE (*Interpretation of the Sphygmogram*).

Hill-Barnard Sphygmometer. *See* PULSE (*Measurement of Arterial Pressure in Man*).

Hill Diarrhœa. *See* SPRUE AND HILL DIARRHŒA (*Hill Diarrhœa*).

Hilton's Method. *See* SUPPURATION (*Acute Circumscribed Abscess, Treatment, Operative*).

Hilum.—The part of an organ at which the vessels and nerves enter, *e.g.* the hilum of the spleen (*hilum lienis*), of the liver (*hilum hepatis*), or of the ovary (*hilum ovarii*).

Himrod's "Cure."—A powder, the fumes of which, when burning, are recommended for the relief of asthma. *See* CANNABIS INDICA; LOBELIA; STRAMONIUM.

Hinckes-Bird's Method of Ventilation.—The admission of air to a room by an interval between the two window sashes, produced by raising the lower one a little, and closing the opening below it by a wooden board on the inner aspect of the window. *See* Ventilation and Warming (*Inlets and Outlets*).

Hindbrain.—Rhombencephalon. *See* Embryology (*Third Week, Fourth Week, etc.*).

Hindgut. *See* Embryology (*Fourth Week, Fifth Week*).

Hindhead. *See* Therapeutics, Health Resorts (*English*).

Hip-Joint.

A. Injuries of.

See also Amputations (*At the Hip-Joint*); Bursæ, Injuries and Diseases of (*Situations of Bursæ*); Circumcision (*Phymosis, Symptoms simulating Hip-Joint Disease*); Deformities (*Congenital Dislocation of Hip*); Deformities (*Coxa Vara*); Fractures (*Pelvis, Femur*); Hysteria, Surgical Aspects of (*Hysterical Affections of Joints, Hip*); Joints, Diseases of; Kidney, Surgical Affections of (*Perinephritis, Diagnosis*); Osteo-Arthropathies (*Arthropathies, Morbid Anatomy*); Rheumatism, Acute (*Symptoms and Course*); Rheumatism in Children (*Symptoms, Arthritis*); Rheumatism, Rheumatoid Arthritis (*Monoarticular*).

Injuries to the hip are numerous and may be serious, for even when they are caused by only slight violence the effects upon the patient are often far-reaching and may be of grave import.

Bruised Hip.—Contusions of the hip, as a result of falls or blows, are usually of importance only in children and in old people. In children because they may form the starting-point of tuberculosis or other infective disease of the joint: in old people because a contusion of the hip often resembles in its symptoms, though never in its signs, a fracture of the neck of the femur. But a fall or repeated blows upon the great trochanter may lead at any age to enlargement of the bursa beneath the insertion of the gluteus maximus, or to a remarkable form of chronic osteoporosis known incorrectly as "interstitial absorption of the neck of the femur."

Symptoms.—In its simpler forms the symptoms of a contusion of the hip differ but little from those of a bruise elsewhere. There is tenderness over the part struck, followed in a few hours by swelling and stiffness, which slowly disappear, whilst the skin passes through a succession of colours due to the oxidation changes produced by the tissues on the hæmoglobin of the extravasated blood. In old people the shock of the injury is sometimes sufficient to paralyse the limb for a time. The thigh lies motionless on its outer side and slightly abducted, whilst the upper part of the thigh is swollen and tender. It is by no means easy to ascertain in these cases whether the patient is suffering from a bruise or from an intracapsular fracture of the femur. In a bruise the thigh

may seem to be slightly lengthened, and the great trochanter maintains its normal relation to the anterior superior spine of the ilium; whilst in a fracture there is some shortening, and the trochanter is displaced and no longer rotates round its proper axis. Even these guides, however, are lost in a patient who suffers from osteoarthritis, and an attempt must then be made to establish the diagnosis by means of a skiagraph. The prognosis of a bruised hip is good, but the practitioner should warn an old person or the mother of a delicate child of certain special risks, which will be described immediately.

Treatment.—The treatment in the simpler forms consists of a few days' rest in bed, during which the hip may be rubbed with an embrocation or may be massaged by a skilful shampooer. When the shock is so great as to make it doubtful whether the injury is a bruise or a fracture the patient must be treated as though he were suffering from an intracapsular fracture.

After-effects.—The pain sometimes continues for a long time, and is increased by exercise after a severe bruise upon the outer side of the thigh in the neighbourhood of the great trochanter. At first there is no shortening, but the patient limps more as the local pain and discomfort diminish, until the injured limb is found to be appreciably shorter than its fellow when the thighs are measured from the anterior superior spines of the ilia to the adductor tubercles. The condition occurs most often in old people, but it is also met with occasionally in young adults who have apparently been healthy until they met with the accident. The pathology is in dispute, and the condition has been described hitherto under the vague expression "interstitial absorption of the neck of the femur." Mr. Arbuthnot Lane has lately advanced good reasons for assuming it to be a result of severe bruising of the cartilages covering the upper part of the head of the femur and the corresponding portion of the acetabulum. The injured cartilages and the underlying bone become absorbed as a result of the chronic inflammation thus set up. The head of the femur becomes altered in shape, and the acetabulum becomes deeper and larger in a manner similar to that seen in osteoarthritis. The great trochanter seems, therefore, as though it had approached the head of the femur, but in reality the trochanter comes nearer to the rim of the acetabulum because the head of the bone lies deeper in its socket, whilst the length of the neck of the femur remains unaltered.

The prognosis is unsatisfactory, especially if the patient is not seen until there is well-marked shortening.

The treatment in the early stages consists in keeping the patient in bed, with a weight and extension applied to the injured limb to remove all pressure from the joint, and to prevent, as far as possible, the continued absorption of the bruised cartilage and bone. When the shortening has commenced, the difference in the length of the legs should be accurately adjusted by a thick-soled boot, for the limping tends to increase the absorption.

Bursal Enlargements.—Blows on the outer side of the hip, injuries, exposure to cold, and certain occupations, such as tailoring, which involve prolonged sitting in bad attitudes, may lead to pain and swelling over the top of the great trochanter. After a longer or shorter time a well-defined swelling appears in the long axis of the limb. The swelling is limited abruptly by the femoral insertion of the gluteus maximus, and is due to an enlargement of the bursa beneath the aponeurosis of this muscle. If it be left untouched it opens at the outer side of the thigh below the level of the great trochanter. In unhealthy people it oftens suppurates and tracks far and wide, leaving most troublesome sinuses, but it is not often associated with any necrosis of the bone even when there has been extensive suppuratioı

Treatment.—The treatment consists in laying open the bursa freely—by cutting through the fascia lata parallel to its fibres—turning out its contents, breaking down all bridges of connective tissue, scraping the wall with a flushing scoop, through which a stream of boracic lotion at a temperature of 110° F. is passing, and afterwards suturing the edges of the wound and obtaining union by first intention. A drainage-tube may be inserted at the time of the operation if it be removed twenty-four hours later. The bursa sometimes communicates by a narrow channel with a small subcutaneous abscess, forming a "collar-stud" abscess, and it is important in dealing with such an abscess not to overlook the point of communication between the small subcutaneous abscess and the larger one situated beneath the fascia lata. The bursa beneath the ilio-psoas tendon (Fig. 1, 10) may also become enlarged as a result of injury or from infection with syphilis, rheumatism, or gonorrhœa. It forms a smooth and fluctuating swelling, which lies below Poupart's ligament, and may extend downwards as far as the middle of the thigh. The swelling may cause neuralgic pains from pressure upon the nerves, or venous thrombosis from pressure upon the deep veins. The movements at the hip are often hampered, and the limb may become slightly flexed, abducted, and rotated outwards. But it is not difficult to distinguish an enlarged ilio-psoas bursa from hip disease, because there is neither shortening, rigidity, nor tenderness of the joint. The treatment consists in freely incising the bursa after it has been exposed through a skin incision. It is absolutely necessary that the wound should heal by first intention. There are other bursæ in the immediate neighbourhood

of the hip-joint situated as is seen in Fig. 1, at the insertion of the ilio-psoas tendon, between the vastus externus and the gluteus maximus, and between the gluteus medius and minimus.

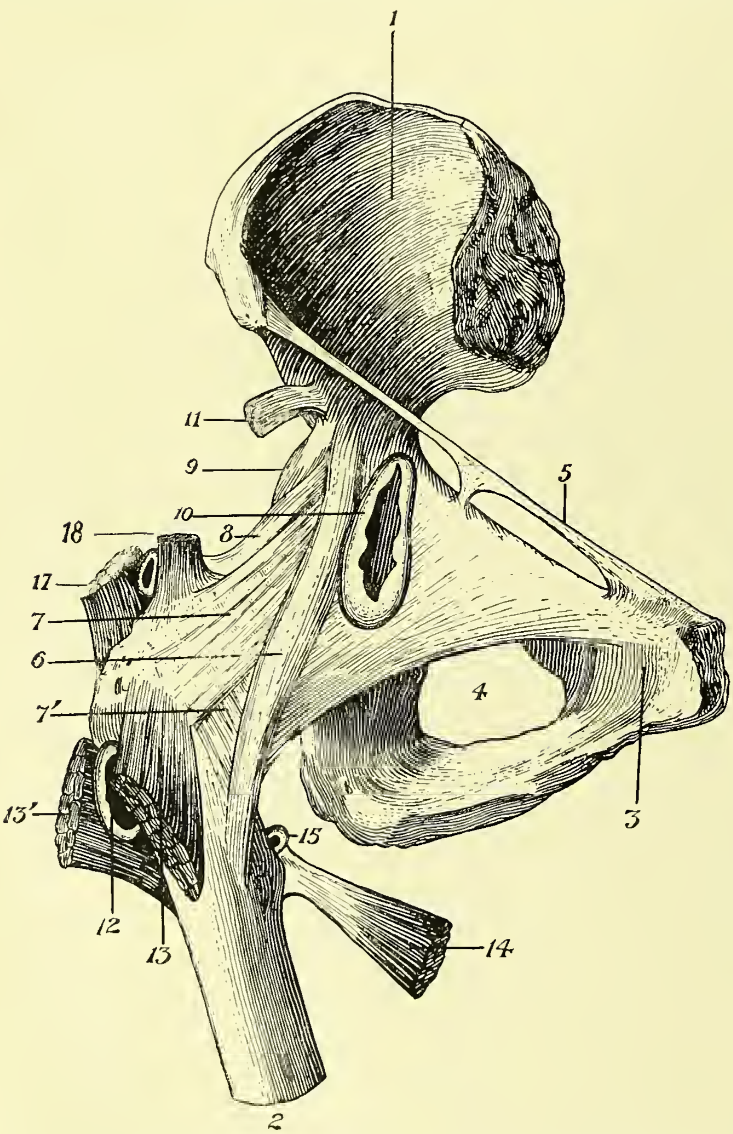


Fig. 1.—Front view of the Hip-Joint. 1. Ilium; 2. Femur; 3. Pubis; 4. The thyroid or obturator foramen; 5. Poupart's ligament; 6, 7, 7′, 8, and 9. The capsular ligament of the hip, divisible into:—6. The inner branch or fasciculus of the ilio-femoral, or Y-shaped ligament of Bigelow; 7. The outer branch or fasciculus of the Y-shaped ligament of Bigelow; 7′. The intermediate portion of the capsular ligament; 8 and 9. The upper portions of the capsular ligament; 10. The bursa lying between the ilio-psoas and the capsule of the joint; 11. The straight head of the rectus femoris cut short; 12. The bursa separating the vastus externus (13) from the gluteus maximus (13′); 14. The tendon of the ilio-psoas cut short; 15. The bursa lying between the ilio-psoas tendon and the lesser trochanter; 16. A small bursa is also shown lying between the insertions of the gluteus medius (17) and minimus (18). (Copied from Testut's *Traité d'Anatomie humaine.*)

I have seen them all enlarged in different persons chiefly as a result of tuberculous disease or osteoarthritis.

Sprained Hip.—Sprains of the hip are generally the result of sudden extension of the thigh combined with abduction, and are rarely caused by forced flexion. They affect the hip in more than one way. Sometimes a few fibres of the pectineus, the adductor brevis, or the adductor longus muscles are torn: in other cases the capsular ligament is wrenched and a little synovitis ensues: whilst in others, again, the ligamentum teres with the fatty tissue at its base is said to be nipped beneath the transverse ligament of the joint. The latter accident happens when a patient slips with the thigh forcibly abducted and at the same time slightly flexed. The thigh becomes everted, abducted, and apparently lengthened, whilst its movements are greatly hampered by the reflex spasm of the surrounding muscles. Flexion followed by rapid extension of the hip sets free the ligamentum teres and restores immediate use to the limb, which should then be kept at rest for a few days. Partial rupture of the muscles and slight inflammation of the synovial membrane are usually amenable to rest in healthy people, but in unhealthy persons they often give rise to much trouble. There may be lameness without any impairment of movement in the joint, perhaps from a little interstitial myositis or from slight teno-synovitis after partial rupture of the muscles or injury to the tendons and their sheaths, or if the synovitis of the hip has been plastic rather than serous, it may have caused local adhesions and thickening of the capsule. These conditions often lead the patient into the hands of the bone-setter, who rightly believes the mischief to be outside rather than inside the joint when there is an absence of pain on direct pressure over the part, or when shocks are impressed upon the heel, particularly if pain is felt upon using the heel as a pivot for the body and when it is produced by stooping forwards. Passive movements and the breaking down of the adhesions, with or without an anæsthetic, are of great service in some of these cases, especially if the surgeon take care to fix any painful spot by pressing his thumb upon it at

the instant when the forcible movement is made.

Wounds.—Wounds of thė hip may so injure the surrounding tissues, and even the capsule, as to lead to serious impairment of the movements of the joint; or they may cause a secondary arthritis; but the joint itself is so thoroughly protected from injury that it is hardly ever injured, except as a result of gunshots. The swelling of the capsule forms the most certain sign of injury, either at once as a result of the effusion of blood, after a short interval from an increase in the synovial fluid, or quite late from suppuration. The swelling is felt easily, and may so raise the femoral artery as to make the pulsations feel subcutaneous. The particular dangers of the injury are liability to suppuration and implication of the pelvic organs. The difficulty of rendering the joint immobile forms a great obstacle to successful treatment, and it is often necessary, therefore, to excise the head of the femur either immediately after the injury or at a later period. The experience gained in the South African war, however, shows that the present gunshot wounds of joints are by no means so serious as those inflicted in previous campaigns. A bullet may pass completely through a joint without leading to permanent impairment of its function.

Dislocations of the Hip are amongst the rarer forms of injury met with in general surgical practice. They are the result of considerable violence, and nearly always occur in adults, though most surgeons have seen a few cases of true traumatic dislocation in children: in old people a fracture of the neck of the femur usually takes the place of dislocation of the hip.

Pathology.—An anatomical examination of the hip shows that dislocation of the head of the femur takes place more easily in some postures than in others, and at the same time it explains why the head of the bone generally takes up certain fixed positions after it has left the acetabulum. The capsule of the hip is strengthened in front by a fibrous expansion—the ilio-femoral or Y-shaped ligament of Bigelow—whilst behind is the ischio-capsular ligament, extending from the back of the tuber ischii to the trochanters. Of these ligaments the ilio-femoral (Fig. 1, 6, 7, 7′) is the more important. It consists of a thickened portion arising from the front of the ilium just below the anterior superior spine, and passing downwards to be inserted into the whole length of the anterior intertrochanteric line; its base extends, therefore, from the front and upper part of the great trochanter obliquely across the front of the femur to just below the lesser trochanter. The outer and the inner parts of the ligament are thicker than the intermediate portion, so that the front part of the ligament consists of two denser fasciculi—the outer and inner branches of the Y-ligament—connected together by a less dense layer. At the back of the joint, in addition to the ischio-capsular ligament, the capsule is strengthened by numerous tendons and muscles (Fig. 2), some of which are in very intimate relation with the capsule, whilst others, from the nature of their tendons, are veritable ligaments. The tendon of the obturator internus muscle (Fig. 2, 5) is one of the most important of these structures, because it forms a compact and well-defined band of fibrous tissue lying immediately over the middle of the head of the femur. Of late years, however, somewhat too great an importance has been attached to it in connection with dislocation of the hip backwards, for it is by no means unusual to find the obturator internus torn.

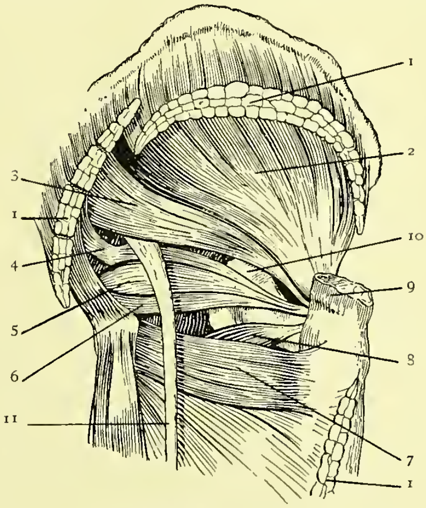


Fig. 2.—The Anatomical Relations of the Hip-Joint. 1. The gluteus maximus cut short; 2. The gluteus minimus; 3. The pyriformis; 4. The gemellus superior; 5. The obturator internus; 6. The gemellus inferior; 7. The quadratus femoris; 8. The obturator externus; 9. The insertion of the gluteus medius; 10. The head of the femur (the capsule of the joint is not shown); 11. The great sciatic nerve. (From Helferich's *Fractures and Dislocations*, translated by Jno. Hutchinson, Esq., Junr. The New Sydenham Society's ed.)

Primary Dislocations.—The capsule is thinnest and least protected opposite the notch in the acetabulum, which is situated at the bottom of the joint and a little behind its vertical axis. The acetabulum, too, is shallow and incomplete at this spot. When the thigh is flexed and abducted the head of the femur presses against this weak part of the joint, and at the same time the ligamentum teres is fully relaxed. A sudden and violent movement imparted to the body of the femur when the thigh is thus bent and abducted may drive the head of the bone out of the socket through a rent in the capsule, the head being displaced downwards and a little backwards, whilst the rent in the capsule varies in size from a minimum equal to the diameter of the head to a laceration involving fully two-thirds of circumference of the acetabulum. This seems to be the usual method

by which a dislocation of the femur is produced so that the common primary dislocation is downwards and inwards. But in some cases a direct dorsal dislocation may be brought about by the application of force to the adducted and flexed thigh. The head of the bone then escapes above the tendon of the obturator internus, if the flexion has only been moderate, below it if extreme. A portion of the rim of the acetabulum is often torn off in direct dorsal dislocation, and in every dislocation a continuance of the force producing the injury may lead to fracture of the pelvis or of the femur. Many of the muscles surrounding the joint (Fig. 2) may be torn, either directly (*e.g.* the obturator externus, quadratus femoris, obturator internus, or pyriformis) by the head of the bone, or, as in the case of the gracilis and adductor longus, by overstretching when there has been extreme abduction. Injury to the great sciatic nerve may lead to neuritis, to partial paralysis, or even to rupture of the nerve.

Secondary Dislocations.—Although in most cases the first position assumed by the head of the bone after it escapes from the acetabulum is downwards, subsequent movements of the injured limb cause this to be only temporary, and the head passes either outwards or inwards, the final position being dependent upon the remnant of untorn capsule binding the shaft of the femur to the rim of the acetabulum. From the character of the capsule the Y-ligament most often remains untorn, and when this is the case, or when it is only slightly injured, the head of the bone passes into certain well-recognised positions, forming a series known as the regular dislocations; but when the Y-ligament (Fig. 1, 6, 7, 7′) is completely or in great part destroyed, the head of the bone may stray into quite unusual places, and a series of irregular or anomalous dislocations is then produced. It should be remembered that although the classification here adopted is useful for clinical purposes, and for guidance in the reduction of the majority of cases of dislocation of the hip, it is by no means pathologically accurate, and it is for this reason that in some cases the ordinary rules for reduction by manipulation fail absolutely.

Regular Dislocations.—In the regular dislocations the head of the femur passes outwards, resting upon the ilium either above the tendon —*the dorsal dislocation* of the older surgeons (Fig. 3, *1*)—or below the tendon of the obturator internus—*the sciatic dislocation* (Fig. 3, *3*). In other cases the head of the femur passes inwards, either inwards and downwards (Fig. 3, *2*) —*the thyroid or obturator dislocation*—or inwards and upwards (Fig. 3, *4*)—*the pubic or subspinous dislocation*—when the head of the bone lies upon the horizontal ramus of the pubes just internal to the anterior inferior spine of the ilium. All inward dislocations present the same general characteristics of abduction of the thigh with rotation outwards, and with a single exception, all outward dislocations cause the thigh to be adducted and rotated inwards. The exception is *the anterior oblique dislocation* when the thigh is everted and crosses the opposite one high up, the toes being turned outwards, the head of the bone lying on the dorsum ilii just behind the anterior inferior spine. The Y-ligament is intact, and the dislocation may be converted into the ordinary dorsal form, in which the thigh is inverted by inward circumduction of the extended limb across the symphysis with inward rotation.

When the outer branch of the Y-ligament (Fig. 1, 7) is torn, the head of the femur may pass forwards and upwards until it lies at a higher level than when the ligament is intact—*a supraspinous dislocation* is then produced—the limb being much shortened, slightly abducted and everted. An anterior oblique dislocation is readily converted into a supraspinous dislocation by rupture of the outer branch of the Y-ligament, so that this variety may be produced in two ways, either by the head passing outwards and upwards or inwards and upwards. When the outer branch of the Y-ligament is torn and the head of the femur passes outwards and backwards, *an everted dorsal dislocation* is produced, the limb being everted and slightly abducted, so that it resembles a fracture of the neck of the femur. Flexion, adduction, and internal rotation of the thigh readily convert an everted dorsal into the ordinary inverted dorsal dislocation.

When the inner branch of the Y-ligament (Fig. 1, 6) is torn, the head of the bone may slip much farther forwards than is usual, and the head may lie upon the pubes close to the pubic spine.

Anomalous Dislocations.—Extensive destruction of the Y-ligament enables the head of the femur to pass upwards or downwards, either vertically below the acetabulum, backwards upon the tuber ischii, or downwards and forwards into the perineum.

Relative Frequency.—The regular dislocations are said to occur in the following order of frequency:—

50 to 55% dorsal above the tendon of the obturator internus.

20 to 25% sciatic or dorsal below the tendon of the obturator internus.

10 to 15% thyroid or obturator dislocations.

5 to 10% pubic or subspinous.

Signs.—The general signs attending a dislocation of the hip are an alteration in the length of the injured limb, diminished power of movement, with locking of the joint in certain positions, and an alteration in the axis of the limb. In a simple dislocation there is no crepitus, and when the displacement is reduced

there is no tendency for the signs to reappear. But it is by no means unusual to find extensive fractures of the pelvis or femur associated with dislocation of the hip, and then, as well as when a portion of the rim of the acetabulum is separ- —drawn from the anterior superior spine of the ilium to the most prominent part of the outer lip of the tuber ischii—and it lies unduly near the iliac spine because the head of the bone is directed backwards upon the dorsum of the

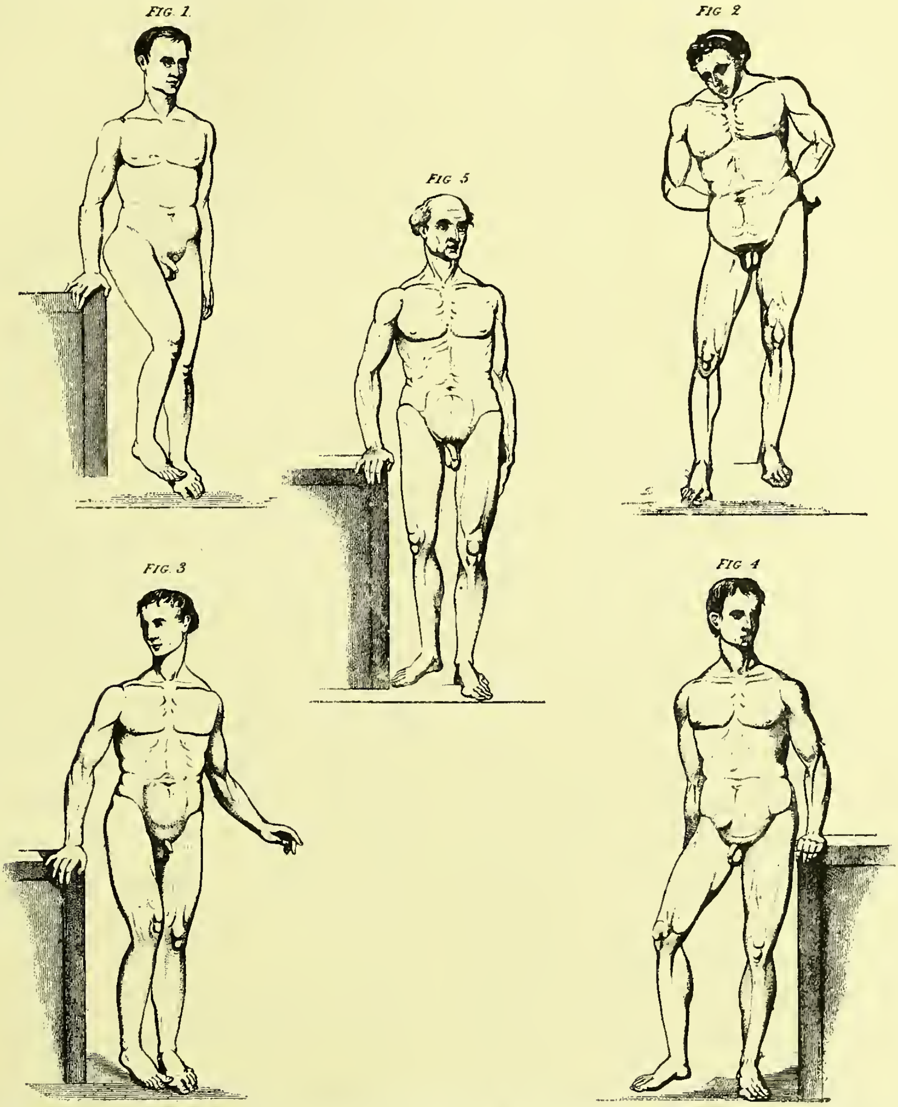


FIG. 3.—Sir Astley Cooper's diagrams to show the positions of the limb in the different dislocations of the thigh-bone, and in intracapsular fracture of the neck of the femur. *Fig. 1.* The right thigh-bone dislocated upwards upon the dorsum ilii. *Fig. 2.* Dislocation of the right femur downwards into the thyroid or obturator foramen. *Fig. 3.* Sciatic dislocation of the right hip. *Fig. 4.* Pubic or subspinous dislocation of the right thigh. *Fig. 5.* Intracapsular fracture of the neck of the right femur.

ated, there will be both crepitus and a tendency for the dislocation to recur after reduction.

The hip is wider and flatter than its fellow in a dorsal dislocation (Fig. 3, *1*) and the injured limb is considerably shorter. The thigh is slightly flexed, adducted, and rotated inwards, so that when the patient is erect the great toe rests upon the instep of the opposite foot, and the knee touches the patella of the uninjured side near the upper margin. The great trochanter lies some distance above Nélaton's line ilium. The femoral vessels have lost their support owing to the displacement of the head of the femur, and the groin appears therefore to be unduly deep. The head of the femur can generally be felt in its new situation, but all the movements of the joint are greatly hampered. Flexion is most free, adduction is difficult, abduction is impossible. When the head of the bone lies below the tendon of the obturator internus (Fig. 3, *3*) there is less shortening of the limb, but the flexion, adduction, and inver-

sion are somewhat more marked than in the dorsal form.

Method of reducing a Backward Dislocation of the Hip.—Reduction is brought about by manipulation, which should be undertaken immediately; but if the patient be not seen until some time after the accident, it is better to defer all manipulation until an anæsthetic can be given, and the attempt made under the most favourable conditions of assistance and position.

The patient lies flat on his back upon a mattress placed on the floor, whilst the surgeon kneels beside him and grasps the leg just above the ankle with one hand, whilst he places his hand, or, if the thigh be heavy, hooks his elbow below the ham of the injured limb. He then flexes the leg upon the thigh to relax the hamstring muscles, and the thigh upon the pelvis to relax the Y-ligament or other portion of untorn capsule, and to bring the head of the femur below the acetabulum. This position of flexion with slight adduction is to be maintained for a few seconds, care being taken not to flex the thigh too much lest the head of the bone pass too far forwards; the thigh is then fully abducted, rotated outwards to its utmost extent, and brought down to the couch in an extended position. The movement of abduction brings the head of the femur from below and behind the acetabulum to the rent in the capsule, the external rotation still further raises the head and causes it to pass through the rent, whilst the extension replaces it at the bottom of the acetabulum.

If reduction does not take place the head of the bone will pass inwards and the dorsal dislocation will be converted into a thyroid or subspinous displacement. The surgeon need not be discouraged, but he should repeat the manipulation, and at the same time he should cautiously increase the amount of adduction and inversion, whilst he diminishes the amount of flexion. He must never allow his efforts to become violent lest he fracture the femur, and he should remember that each repetition of the manipulation is likely to lead to further tearing of the soft tissues around the joint. When the rent in the capsule is small, as is shown by the restricted movements of which the limb is capable, circumduction will sometimes restore the head of the bone to its socket, for the capsule is then so short that the movement compels the head to hug the rim of the acetabulum until it re-enters the cavity. The circumduction is effected by sweeping the leg and thigh round in a wide circle, the patella being always kept to the front, for the articular surface of the head looks nearly in the same direction as the internal condyle of the femur. This method of manipulation, however, is open to two serious objections. It happens sometimes, after an apparently successful reduction of a dorsal dislocation by free circumduction, that the knee remains bent whilst the thigh continues flexed on the pelvis and is abducted and rotated outwards. This want of freedom is due to the fact that the sciatic nerve is stretched over the front of the neck of the femur, lying first beneath the inner portion of the capsule and the tendon of the psoas, and then descending through the rent in the muscular wall of the thigh to the adductor magnus. A second objection to the method of reduction by circumduction is that a part of the capsule may become folded over the head of the bone and may thus prevent reduction.

In the thyroid or obturator dislocation (Fig. 3, *2*) the injured thigh is longer than the other, though too much reliance must not be placed upon this sign; it is abducted and flexed. The hip is flattened and the great trochanter is less prominent than normal. The toes point forwards. When the head of the bone lies in the perineum there is marked flexion and abduction of the thigh.

Reduction is accomplished by placing the patient supine as before. The surgeon then flexes the leg on the thigh, the thigh on the abdomen, adducts, inverts, and brings the thigh downwards in extension. This form is usually easier to reduce than the dorsal displacement.

In the pubic dislocation (Fig. 3, *4*) the hip is flattened and the great trochanter lies above Nélaton's line and nearer to the symphysis pubis than is natural. The thigh is shortened, abducted, rotated outwards, and slightly flexed. The femoral artery is displaced inwards and the head of the bone can be felt in its new position. This form of displacement is sometimes attended by great pain in the course of the anterior crural nerve.

Reduction is brought about in the same manner as in the obturator variety by flexing the leg on the thigh, the thigh on the abdomen, then adduct, invert, and bring down in full extension.[1]

The after-treatment of a reduced dislocation consists in keeping the patient in bed for a fortnight, the injured limb being placed between

[1] Mr. Sturrock has recently (*The British Medical Journal*, i. 1900, p. 845) reduced two regular dislocations of the hip by the following manipulation: The patient being anæsthetised and laid upon his back on the floor, the surgeon kneels upon his left knee when the left hip is dislocated, and on the left side of the patient. The patient's thigh is then carefully flexed to a right angle, and while this is being done the leg is also flexed to a right angle and laid with the most prominent part of the calf on the right knee of the surgeon. The ankle is then firmly grasped with the left hand and the condyles of the femur with the right. The thigh is then abducted for thyroid dislocations, adducted for dorsal and pubic, and rotated inwards for all varieties, by drawing the foot away from the middle line and keeping the knee steady. Traction is now made by steadily depressing the ankle, the surgeon using his knee as the fulcrum: the patient's leg makes a most powerful lever. The thigh is then rotated outwards, and, while this is being done, the head of the femur slips into the acetabulum.

sandbags or bandaged to a long outside splint. Passive movement may then be employed, but the patient should not be allowed to walk about for at least six weeks after the accident, though he may be permitted the use of crutches. The duration of treatment in a case of dislocation complicated by fracture will necessarily be prolonged, and a weight and extension should be applied.

The prognosis after reduction will depend upon the nature of the injury and the ease with which the displacement has been reduced. It is good in simple cases, but the patient should be warned that the accident may recur, that there may be some permanent lameness from injury to the muscles, that there may be some neuritis, and that it may form the starting-point of disease of the joint.

The obstacles to reduction are numerous and are sometimes insuperable. One of the most common appears to be due to the fact that the capsule has been torn from the neck of the femur, so that it hangs as a covering over the acetabulum and prevents the head re-entering the socket: in other cases part of the obturator externus is torn, and the muscular fibres are pushed before the head into the acetabulum, whilst in yet other cases the head of the femur may lie as it were "in chancery" between the tendons of the pyriformis and the obturator internus muscles (Fig. 2, 3 and 5). When the capsule has been pushed into the socket from the dorsal aspect it may sometimes be removed by flexing and abducting the thigh, whilst if it has entered from the thyroid aspect flexion and adduction of the femur may displace it. After engaging the inverted capsule the femur should be rotated inwards to tighten the Y-ligament and drive the head well into the acetabulum, whilst at the same time the knee is raised and the capsule or muscle is thus removed from its cavity.

The irreducibility in long-standing dislocations is due to causes other than those just mentioned, though the capsule again plays an important part. When the cavity of the acetabulum remains unoccupied the torn capsular ligament falls over it, and the stump of the ligamentum teres undergoes inflammatory changes. A proliferation of connective-tissue cells takes place, and the socket becomes occupied with connective tissue which is at first soft, but soon becomes dense and fibrous. If the rent in the capsule is only a small one it often heals early, and has been found completely repaired eighteen days after the dislocation. The ligament teres, too, shows remarkable powers of repair, and a new and very complete joint may be formed at the place where the head lies in its new position. The shortening of the muscles and tendons inserted into the upper end of the femur and fibrous adhesions, or a new capsule formed about the head, greatly militate against the successful reduction of old-standing dislocations of the hip.

The treatment of unreduced dislocations of long standing is one which may require the most anxious consideration of the surgeon. It consists necessarily in leaving the patient alone, in subcutaneous division of the contracted muscles and tendons, in osteotomy, in arthrotomy, or in excision of the head of the femur. When the inconvenience is slight and the dislocation of long standing it is generally wise to leave the patient alone, but when he is crippled and in pain, arthrotomy and excision of the head of the bone seem at present to offer the most satisfactory results. Arthrotomy consists in cutting down upon the acetabulum by an incision planned to permit easy access to the great trochanter. In the majority of cases this will be found to be in front of the joint along a line separating the posterior border of the tensor fasciæ femoris from the gluteus medius. The head and neck of the femur are then laid bare, and all the muscular attachments are divided as low as the lesser trochanter, until the whole upper end of the bone, including the great trochanter, is absolutely free, the division being subperiosteal as far as is possible, and the proximity of the great sciatic nerve not being overlooked. The acetabulum is then excavated and enlarged sufficiently to allow the head of the femur a ready entrance. The head is replaced in the socket thus prepared to receive it, the wound is closed, extension is applied, and the limb is fixed for at least a month in a position of slight abduction. Excision should only be performed when arthrotomy has failed, or is manifestly impossible, for though it gives the patient a useful limb it also gives him a shortened one. When an excision has been decided upon, therefore, as little of the neck as possible should be removed.

Other difficulties may also arise in connection with a dislocation of the hip, and chief amongst these are the cases where the dislocation is compound or when the femur is broken in the upper third. In the less serious cases of compound dislocation the wound and the surrounding skin should be thoroughly cleansed, and the head of the bone should then be guided into the socket, the wound being enlarged if it be necessary, but more often it will be necessary to excise the head of the bone. Amputation is so fatal an operation that it should be avoided except as a very last resource. But when it has to be done, the operation should not be delayed until inflammation and suppuration have made all the tissues highly vascular, as hæmorrhage is the chief danger of this operation. When the dislocation is complicated by a fracture, an incision must be made along such a line as will enable the head to be replaced and the fracture to be united with stout silver wire. The bulk of the soft tissues covering the injured hip will sometimes render

impracticable any operation, and the surgeon must then be content to leave the dislocation unreduced and secure union of the fracture. The subsequent treatment will depend upon the amount of movement which the patient obtains.

Epiphyseal Separations.—*The anterior inferior spine of the ilium* has attached to it the straight head of the rectus femoris muscle, which is rendered taut when the hip is extended, the reflected head being tightened when the rectus is used to extend the knee during flexion of the hip. Separation of the epiphysis of the anterior inferior spine of the ilium is the result of muscular force transmitted through the straight head of the rectus, and has occurred in young adults who were just bounding off at the beginning of a race, when they had been standing with one leg advanced and bent both at the hip and knee whilst the injured leg was straight. The symptoms were sudden but not very severe pain, flexion at the hip and knee with crepitus, sometimes difficult to obtain, as only a scale of bone may be detached. The treatment consists in the application of a plaster of Paris spica for a month. The prognosis is good.

The epiphyseal head of the femur may be separated at any time after the age of 4 years, though the accident is most common between the ages of 14 and 18 years. It is probable that in early infancy the whole upper end of the femur may be detached from the shaft, but in older children it is an intracapsular separation of the cartilaginous head. The accident happens as a result of direct injury to the outer side of the thigh below the great trochanter when the leg is adducted and semiflexed. The injured limb is everted owing to the weight of the leg, and there is slight flexion with abduction or adduction. The eversion is sometimes replaced by inversion, due perhaps to the lower fragment being placed in front of the upper part of the bone. The great trochanter is more prominent, and lies higher on the injured than on the uninjured side, and when the separation is complete there is muffled crepitus with very considerable shortening. The amount of movement varies with the separation of the fragments; it is usually slight, but when the periosteum remains untorn the patient may retain considerable power over his leg, for the periosteum covering a growing bone is relatively thicker and stronger than that covering a mature bone. The pain varies greatly, but it seems to be somewhat less severe than in a fracture of the neck of the femur.

The injury has to be distinguished from a traumatic and from a congenital dislocation, from a fracture of the neck of the femur, from hip disease, and from a simple bruise. The Röntgen rays help materially in the diagnosis.

The prognosis is generally good. Bony union occurs unless wide separation with laceration of the periosteum leads to non-union, an unfortunate result, which is most likely to occur in children who have been allowed to move the limb unrestrictedly in consequence of failure to recognise the nature of the injury. Some impairment of growth may take place in the upper third of the femur, leading to alterations in the head and neck of the bone, simulating the rachitic deformity known as coxa vara (see p. 229).

The treatment consists in the application of a weight and extension for three weeks, after which the patient is allowed to go about on crutches either in a Thomas splint or with a poroplastic splint fitted round the pelvis and extending down the outer side of the thigh. The prognosis is good though the repair is sometimes slow.

The epiphysis of the great trochanter may be separated between the thirteenth and sixteenth years of age generally by direct violence, as by a fall on the hip or a blow on the great trochanter, though the injury is not necessarily severe. In many cases the displacement is slight, but when the separation is complete the epiphysis is drawn upwards and somewhat backwards and inwards by the glutei and rotator muscles. Crepitus may or may not be felt, but there is always pain, swelling, and extravasation over the seat of injury. The prognosis is by no means good, for several of the recorded cases have died of septic poisoning. A long external splint, interrupted at the hip, should be applied as soon as possible, and the patient must be carefully examined from day to day to discover whether he has any symptoms of infective osteomyelitis. If necessary an incision should be made down to the bone before an abscess forms.

Fractures of the Hip.—*The acetabulum* may be broken either through its cavity or along the rim. A fall upon the trochanter may cause a simple fissure extending across the socket, it may split the acetabulum into its three constituent parts, or it may drive the head of the femur through the base into the pelvis. Fissured fractures give rise to few symptoms besides pain and lameness, and even when there has been considerable displacement of the fragments in the more severe fractures most of the recorded cases have been diagnosed as examples of fracture of the neck of the femur. Such errors will probably be less frequent in the future, as the Röntgen rays can be utilised when there is any doubt as to the nature of the injury. When the head of the bone is driven into the pelvis, pressure upon the great trochanter causes deep-seated pain, and there is often evidence that the pelvic viscera, especially the bladder, have sustained serious injury.

The prognosis of fissured fractures of the acetabulum is favourable, but when the head of the bone has been driven through the base the injury often proves fatal, either at once from shock or after a time from prolonged suppuration and septic absorption.

In the simpler forms of injury the treatment

consists in keeping the patient at rest with a pelvic band and a weight and extension on the injured side. This may be replaced by a plaster of Paris spica at the end of six weeks, after which the patient is allowed to go about on crutches.

Fractures of the rim of the acetabulum are usually associated with direct dorsal dislocations of the femur. The symptoms are those of a dislocation of the hip backwards with the addition of crepitus and a great tendency for the displacement to recur after reduction. The prognosis is not very good since permanent lameness may occur from the impossibility of keeping the head of the femur in the acetabulum. The treatment consists in reducing the dislocation, and afterwards applying a Hodgen's splint (vol. iii. p. 360 and Fig. 4) for a month or six weeks after the accident.

Fractures of the Upper End of the Femur.—Although *intracapsular fractures* of the neck of the femur generally occur in persons over 60 years of age, they are undoubtedly met with as rare accidents in children in consequence of injuries of the same nature but of somewhat greater severity than those causing a separation of the epiphysis. The fracture is common in old people, because the changes which occur in the bones as a result of increasing age are especially marked in the upper third of the femur. These atrophic changes lead to an enlargement of the cancelli in the neck of the femur, whilst the compact layers of bone undergo a process of absorption which renders them particularly liable to yield when they are subjected to a strain or twist. The osteoporosis is especially marked in the denser layer of bone which reaches from the under part of the head to the lesser trochanter—known as the *calcar femorale* of Merkel. In women, too, the neck of the femur has to withstand a greater strain in supporting the weight of the trunk than in men, for in women the width of the haunch-bones causes the neck of the femur to form with the shaft an angle which is nearer a right angle than it is in the male pelvis.

An intracapsular fracture, therefore, often occurs in persons over 60 years of age, and in women more often than in men. Falls upon the feet or knees, and blows upon the front of the great trochanter, are particularly likely to produce an intracapsular fracture, and it is often remarkable how slight an injury, especially if a twisting force be applied, is sufficient to cause it. The signs (Fig. 3, *3*) are eversion of the limb with slight shortening. The great trochanter is less prominent, lies higher, and is situated more posteriorly on the injured than on the uninjured side. In a few cases the eversion is replaced by inversion. Crepitus may be obtained by drawing the thigh downwards and rotating it inwards, but it is not usually well marked, nor is it necessary to obtain it. The fascia lata is relaxed (Fig. 5) between the great trochanter and the crest of the ilium in consequence of the loss of resistance which is normally offered through the neck of the femur. Pain of a starting character is referred to the insertion of the psoas, and may extend down the thigh to the knee; it is increased by adduction and flexion of the hip. There is usually much loss of power over the limb, but when the reflected portion of the capsule is untorn the patient may be able to stand upon the injured limb. The experiment, however, is a dangerous one, and should never be tried, because so long as the two fragments are held in apposition there is some chance of obtaining bony union. Impaction rarely takes place, but when it occurs the neck is driven into the head of the femur. The prognosis is most unsatisfactory, both as regards the life of the patient and the use of the limb. Old persons with feeble circulations and atrophied lung tissues are liable to suffer from serious hypostatic congestion when they are suddenly compelled to lie flat upon their backs in bed. The temperature rises, there is some bronchitis, a little wandering of the mind, and in a few days, unless care be taken, and often when

Fig. 4.—A photograph of a case of fractured femur treated by a Hodgen splint to show the method of suspension. The details of the splint may be seen in vol. iii. p. 360. (From a photograph by Mr. G. S. Wynne of St. Bartholomew's Hospital.)

every care is taken, the patient dies: in other cases bedsores are a fertile source of trouble. In old people non-union is the rule in cases of intracapsular fracture. The non-union is either complete or fibrous, but bony union occurs sufficiently often to cause the surgeon to make

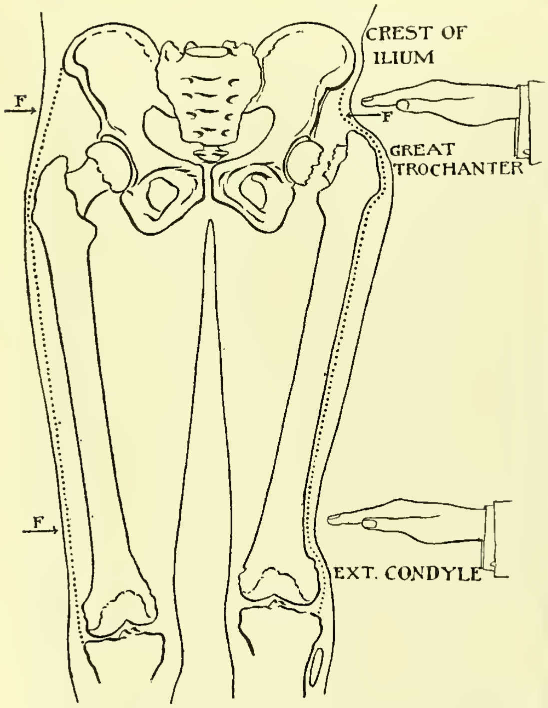


Fig. 5.—Diagram to show the manner in which the ilio-tibial band becomes relaxed in an intracapsular fracture of the femur. The diminished resistance of the ilio-tibial band on the affected side sometimes affords important corroborative evidence of the nature of the injury in doubtful cases. (From Allis's Gross Prize Essay.)

every attempt to secure it unless the constitutional symptoms are so serious as to render it necessary to save life at the expense of the utility of the limb.

The treatment consists in applying an extension (vol. iii. p. 361) with a light weight of 2-3 lbs. to steady the limb and prevent the starting pains. A sandbag is placed on either side of the injured limb, and the patient is kept in bed for a month. A Thomas splint is then fitted, and the patient is allowed to go about on crutches. If bedsores or any symptoms of bronchitis appear, the patient should be put into a Thomas splint at once and allowed to get up.

The measurements required by the instrument maker for the splint are as follows: the circumference of the chest at the line of the nipples; the circumference of the thigh two inches below the fold of the buttock; the circumference of the lower third of the leg about three inches above the malleoli. The length from the line of the nipples to two inches below the fold of the buttock; the length from the line of the nipple to the lower third of the leg about three inches above the malleoli. It must be stated for which side of the body the splint is needed, and if crutches are required, the distance from the axilla to the sole of the foot on the sound side must also be noted. A boot for the sound limb must be sent that a pattern may be fixed to it. The price of a single Thomas splint varies from 6s. 6d. to about a guinea and a half.

Fractures of the neck of the femur occur in younger persons than the majority of intracapsular fractures. They are often known as extracapsular fractures, but incorrectly, because the capsule of the hip is attached in front to the intertrochanteric line of the femur, and the line of fracture is therefore only outside the femur posteriorly. These fractures are the result of direct and serious injury, such as falls upon the outer side of the great trochanter. The back of the neck of the femur yields first, and is often comminuted, the rest of the neck then breaks, and the proximal end of the bone is driven into the great trochanter. If the force is expanded the fracture remains impacted, but if it continue the bone is further split, and the trochanters are either detached or the great trochanter is so much comminuted as to set free the impacted neck of the femur. The fracture is not found more often in women than in men, as is the case in intracapsular fractures, and there is usually great pain made worse by pressure over the great trochanter. The limb is generally powerless, but the patient has control over it more frequently than is the case after an intracapsular fracture, and when the impaction is complete a few steps may even be taken with the injured limb.

The signs consist in a shortening, which, though slight at first, may suddenly become considerable owing to the separation of the impacted fragments. The great trochanter is increased in size directly after the accident; it lies nearer to the anterior superior spine of the ilium, and the hollow of the groin becomes filled up. The limb is generally everted owing to the posterior part of the neck of the femur being less resistant than the anterior portion, but a few cases of inversion have been observed. The ecchymosis and bruising are considerable.

The prognosis is good because firm bony union occurs in a comparatively short space of time. The impaction should be allowed to remain in people over middle age, who will then walk with a limp, but in young and vigorous adults the two fragments should be disengaged, in the hope that the union may take place without any shortening.

The treatment consists in extending and supporting the limbs by means of a Hodgen splint, bent at the knee to an angle of 130°. The leg is first washed, dried, dusted with zinc oxide, and shaved if necessary. An extension apparatus of moleskin strapping with a wooden

stirrup is secured to the leg and lower part of the thigh in the manner described (vol. iii. p. 361). Strips of house flannel are cut and arranged beneath the limb, from the great trochanter to the ankle, each strip overlapping the one above it, and each being three inches broad, and rather more in length than the circumference of the limb at the part it is to overlap. The splint is then placed over the limb so that the upper cross bar lies an inch below Poupart's ligament, and the lower cross bar is over the dorsum of the foot on a level with the instep, whilst the outer bar is as high as the anterior superior spine of the ilium, and the inner bar is opposite the tendon of the adductor longus. The strips of flannel are then pinned to the sides of the splint in order, and from above downwards, until the thigh and leg lie in a trough. A stout piece of blind cord is then attached to the stirrup of the extension, and to the end of the splint which projects at least five inches beyond the foot. The limb is slung by means of a pulley and cord attached to the splint by two pairs of hooks soldered to the sides, and the cord with a weight at the end runs over a pulley in a standard placed at the foot of the bed (Fig. 4). The position of the limb required to bring the two fragments into good apposition can only be determined by experiment, but the limb, even to its extreme upper limit, should lie free of the bed, and at the lower end the splint should be at least ten inches above it. The piece of cord attaching the stirrup to the end of the splint is a valuable test of the efficiency of the apparatus; so long as it remains taut good extension is being exercised upon the limb through the agency of the splint, but if it become slack the appliance must be readjusted. When a Hodgen splint is not available a long outside splint with a weight and extension may be applied for six weeks, and the patient may then be allowed to go about on crutches and in a Thomas splint for a further period of three to six weeks, the actual time depending upon his age and weight.

Fracture of the great trochanter is a rare form of fracture resulting from direct injury to the hip. The symptoms are flexion, deformity, limitation of movement. The great trochanter is drawn upwards and lies above Nélaton's line. It appears in some cases as if an actual increase had taken place in the length of the limb, and the accident is mistaken for a dislocation of the head of the femur, especially when the patient is only seen some time after the injury. Bony union generally occurs with the great trochanter in its new position, and the patient therefore limps after the accident. It is sometimes worth while to pin the great trochanter to the shaft, but in most cases the treatment consists in keeping the patient in bed with his limb between sandbags or in a Thomas hip splint.

Coxa vara is a name given to a rickety incurvation of the neck of one or both femora which is seen more often in growing children than during the early years of life. Particular attention was first called to it in 1889 by E. Müller, who said that "in adolescence without apparent cause or following a slight injury, the patient begins to limp and complain of fatigue and pain about the affected joint on exertion. Shortening of the limb is soon apparent and is caused by elevation of the trochanter above Nélaton's line. The limb is usually extended or flexed to a few degrees and somewhat rotated outwards. The motion of the joint is slightly diminished, particularly in abduction. There is no local tenderness on pressure." Coxa vara occurs in those who carry weights, do much walking, or are subject in other ways to prolonged fatigue. Its onset is marked by a peculiar stiffness of the hip referred to "growing pains," the stiffness being worse on rising after sitting for a time. It is relieved by complete rest. The shortening may be considerable, amounting to as much as $1\frac{1}{2}$ inch. The prominence of the trochanters is especially well seen by flexing the thighs, and the displacement of the trochanter in relation to Nélaton's line is backwards as well as upwards. This is usually associated with external rotation of the limb, but in some cases there may be external rotation without any upward displacement. Tilting of the pelvis with consecutive scoliosis and secondary genu valgum or knock-knee may result from the deformity.

The diagnosis, *prognosis*, and *treatment* are discussed in article "Deformities," vol. ii. p. 296.

B. Diseases of.

See also DEFORMITIES (for Congenital Dislocations).

In individuals of average nutrition the constituent parts of the hip-joint are not available for direct examination. We are unable to detect the presence of fluid or the existence of synovial thickening so readily as we can in other joints, so that in the diagnosis of hip disease we are compelled to rely upon evidence which is indirect, *e.g.* the presence of a limp in walking, an alteration in the attitude of the affected limb, restriction or loss of the normal movements of the joint, etc. The accurate interpretation of these phenomena necessitates considerable clinical experience, and no little care, on the part of the observer. He must be able to measure correctly the length of the lower limbs and of their different segments, to ascertain the position of the great trochanter in relation to Nélaton's line, to Bryant's triangle, and to determine the attitude of the lower limbs in relation to the pelvis, and of the pelvis in relation to the spinal column.

The hip-joint may be located as follows:—From the mid-point of a line drawn between the anterior superior iliac spine and the symphysis pubis a perpendicular line directed downwards will divide the joint into two equal halves. A horizontal line projected inwards from the upper edge of the trochanter will pass through the centre of the head of the femur; in children the trochanter is at a higher level, so that a similar line will touch the highest part of the head. The whole of the anterior and fully one-half of the posterior aspect of the neck of the femur is covered by synovial membrane, so that not only lesions of the epiphysis and epiphysial junction, but also those in the neck of the bone, are capable of direct extension to the synovial membrane and to the cavity of the joint.

While the capsular ligament of the hip is of extraordinary strength, there are certain weak points through which infective material may escape from the joint into the surrounding tissues; the most important of these is on the anterior aspect, where a bursa intervenes between the capsule and the ilio-psoas; the bursa communicates with the joint in one out of ten bodies; a second weak point exists immediately above the trochanter minor, where the tendon of the obturator externus passes above the insertion of the ilio-psoas; a bursa may intervene between these structures, and may communicate with the joint.

When the spongy bone constituting the floor of the acetabulum is the seat of infective disease the latter may extend within the pelvis, giving rise to a swelling which may be felt per rectum, and it may be also to complications involving the pelvic viscera.

Only these diseases of the hip are here described which present distinctive features. For other diseases the reader is referred to the general article on "Diseases of Joints."

TUBERCULOSIS. MORBUS COXÆ.

Tuberculous disease of the hip, or "hip disease" as it is commonly called, without further qualification, is especially common in the class of patients who frequent hospitals. It is of great importance, inasmuch as it is a cause of prolonged invalidism or confinement to bed; it is attended with a considerable mortality (only second, so far as the skeleton is concerned, to disease of the spine and pelvis), and, especially if untreated, it may result in permanent deformity. The clinical study of hip disease is not unattended with difficulty, partly because the morbid changes are less open to inspection and palpation than is the case in other joints, and partly because of the complicated relationships of the lower extremity to the pelvis and spine in the various phases of the disease.

GENERAL FACTS AS TO AGE, SEX, RELATIVE FREQUENCY, ETC.—It is essentially a disease of early life; it rarely commences after puberty, and very rarely after maturity. In any case of hip disease in an adult one should be doubtful of its tuberculous origin, unless it is a relapse of antecedent disease.

Hip disease is slightly more common in the male than in the female.

The frequency of disease at the hip, in relation to that of other parts of the skeleton, varies

with age; in children it comes second after the spine; in adolescents it comes third after the spine and knee.

In a certain number of cases hip disease is primary in the sense that it is the only discoverable tuberculous lesion; in others it follows upon tuberculosis of the lung, kidney, lymphatic glands, etc., and is thus to be regarded as definitely metastatic and less favourable.

When it develops after an injury the patient is to be regarded as already tuberculous, and the injury merely plays the part of a localising factor.

Pathological Anatomy.—This will only be considered in so far as it affords an explanation of certain of the clinical features, or affords guidance in diagnosis and treatment.

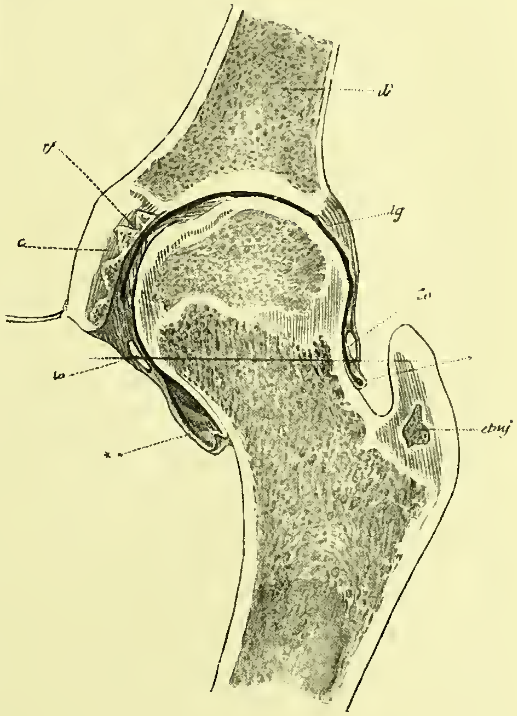
I. *The relative frequency of disease in the synovial membrane and in the bones* has an important bearing on prognosis; while recovery under conservative treatment may be reasonably anticipated in disease of synovial origin, operative measures will be required in the majority of cases in which there are lesions in the bones.

Observations based on specimens removed by operation or preserved in museums are misleading, as these are derived from the graver and more persistent forms of the disease. The general experience, however, is unanimous that bone lesions bulk more largely in the pathology of hip disease than is the case in other joints. The most recent estimate is that given by Krause based on 177 cases; the origin of the disease was osseous in 129, synovial in 26, and doubtful in 22. The proportion of osseous disease would appear to be even higher in the case of children; synovial disease *per se* is less infrequent in young adults, and in cases where other joints are affected simultaneously with the hip.

II. *Seat of the Bone Lesions.*—The acetabulum and upper end of the femur are affected in about equal proportion. The most frequent site *in the femur* is at or near the junction of the epiphysis and neck of the bone; in this situation the tuberculous material has only to erupt through the cortex of the bone in order to infect the synovial membrane or the entire joint. Gross lesions at the epiphysial junction may cause detachment of the epiphysis, and the latter may subsequently become fixed in the acetabulum. Lesions in the epiphysis itself are on a smaller scale. Those at the base of the neck and in the trochanter, although capable of running an extra-articular course, are, if left to themselves, liable to extend to the joint; in exceptional cases they may do so by tunnelling the cervix and head of the bone. *In the acetabulum* the wall or the floor may be the original site of the disease; in either case the joint can scarcely escape infection, and the latter is usually more serious than when it arises in the femur.

III. *The nature of the bone lesions* has a considerable influence on the clinical features of hip disease; we may distinguish between the primary lesions, which are essentially tuberculous, and the secondary lesions, which are largely the result of pressure.

The tuberculous lesions present the same characters and wide variation as in other joints; it should be noted, however, that the sclerosed foci, which result in necrosis, are



comparatively frequent, especially in the femur, where they are usually wedge-shaped, the base of the wedge abutting on the epiphysial disc or on the articular surface of the head. Riedel estimated that sequestra are present in 70 per cent of the cases subjected to operation. Disease in the floor of the acetabulum may result in perforation at one or at several points, and in the formation of an abscess within the pelvis. Gross alterations on the pelvic aspect of the acetabulum may be discovered on rectal examination.

The secondary changes from pressure involve an alteration in the size and shape of the bones subsequent to the destruction of the articular cartilage. While the disappearance of the surface bone may be the direct result of the tuberculous process, the more extensive alterations, at present under consideration, are the result of the absorption of bone, already softened by inflammation (rarefying ostitis), under the influence of pressure. In untreated cases, the bones, being immobilised in a definite position by muscular contraction, exercise a continuous

and injurious pressure upon one another. *The head of the femur* undergoes absorption from above downwards; it loses its spherical shape, becomes flattened or truncated, or may disappear altogether; the neck of the bone in its turn may be absorbed. In the *acetabulum* the bone is absorbed in an upward and backward direction under the influence of the pressure exerted by the head of the femur; the socket is thereby enlarged and elongated towards the dorsum ilii; by active formation of new bone an attempt is made to maintain a buttress for the head of the femur; but the further progress of the disease may result in a further absorption of bone and a still greater enlargement of the socket; the lower and anterior part of the acetabulum, which is no longer in contact with the head of the femur, is filled up with granulation tissue, which may or may not be tuberculous. To this progressive enlargement and displacement of the socket Volkmann gave the suggestive name of "*wandering acetabulum.*" Its clinical importance is obvious, since the resulting displacement of the femur is the most important cause of actual shortening in advanced, and especially in untreated cases of hip disease; the displacement may be so considerable as to simulate a dorsal dislocation; the prominent posterior edge of the enlarged and displaced socket may be felt as a hard swelling above and behind the great trochanter.

Among the secondary results of tuberculous disease at the hip special mention must be made of *the occurrence of dislocation*; this may result from the alterations in the bones already described, or from destruction or softening of the capsular and round ligaments, or from a combination of these; the displacement of the head of the bone may be further assisted if the acetabulum has been largely filled up with granulation tissue. The direction of the displacement is nearly always upwards and backwards; the dislocation may be complete on to the dorsum ilii, or it may be incomplete; in the latter case the head rests on the upper and posterior edge of the acetabulum, and presents a deep groove corresponding to the line of contact.

The formation of abscess, which is such a frequent accompaniment of hip disease, usually proceeds from the joint itself, and the abscess usually communicates with the joint through an opening in the capsule. The abscesses within the pelvis usually communicate with the joint through the acetabulum. Abscesses may form in the vicinity of the joint from an extra-articular focus of tubercle at the base of the neck or in the great trochanter.

Clinical Features.—For convenience of description it is customary to divide these into stages, although never sharply defined from each other nor present in every case of the disease. There is usually a well-marked stage of onset characterised by a voluntary limp, by pain, and by restriction of the movements of the joint; a second stage with abduction of the limb and apparent lengthening, and a third stage with adduction and apparent or actual shortening. Of these stages, the first may be absent or may escape observation, the second is very typical of hip disease, but may also be absent, inasmuch as cases are met with of an acute type in which the attitude from the outset is one of adduction. The third stage is the one most often absent, either because the patient has recovered before reaching it, or because the adduction by which it is characterised has been prevented by treatment. The formation of abscess may take place at any stage in the progress of hip disease.

Initial Stage. — This probably corresponds either to disease which is still confined to the synovial membrane or to a lesion in the bone which has not yet opened freely into the cavity of the joint. The onset of hip disease is usually so insidious that the patient or his friends are rarely able to define the date on which it commenced. If injury is alleged as an exciting cause, some weeks usually elapse between the receipt of the injury and the onset of symptoms. In the case of a child it is usually brought for advice because it has begun to limp. The statement may be made that it has become pale, that it has ceased to take its food well, that its sleep has been disturbed, and that the pain and the limp, after coming and going for a time, have become more continuous and pronounced. On asking the child to walk, it may be observed that the affected limb is dragged in such a way as to avoid movement of the hip, and to substitute for this, movement of the pelvis; the child rests the weight of the trunk on the affected limb as little as possible, and inclines to rest on the balls of the toes rather than on the heel. There is usually some wasting of the muscles of the thigh and slight flattening of the buttock. Diminution or loss of the gluteal fold indicates a degree of flexion at the hip which might otherwise escape notice. Pain is complained of in the hip or shooting down to the inner side of the knee (through the obturator nerve); sometimes the pain is confined to the knee, and if the practitioner restrict his examination to this joint he may overlook the disease at the hip. At this early stage the attitude of the limb is not constant; it may be quite natural at one time and slightly flexed or abducted at another. Tenderness of the joint is of little diagnostic importance; it should be looked for either in front or behind the head of the bone; complaint of pain on driving the head against acetabulum may occasionally assist in the recognition of hip disease, but the diagnostic value of the procedure has been overrated. Most stress is to be laid on *testing the functions*

of the joint, and if it is carried out gently and without jerking there is no complaint of pain. The movements should be practised on the sound limb in the first instance in order to reassure the child that no violence is contemplated. The child should lie on its back, either on its mother's knee or on a table. On slowly flexing the thigh of the affected limb, it will be found that the range of flexion at the hip is soon exhausted, and that any further flexion takes place at the lumbo-sacral junction. The child is then made to lie on its face with the knees flexed in order to test the movements of rotation ; the thigh is rotated outwards and inwards, and on comparing the two sides it will be found that rotation is restricted or abolished on the side affected, any apparent rotation taking place at the lumbo-sacral junction. These tests, as they may be called, reveal the presence of *rigidity* which results from the involuntary contraction of muscles ; it is the most reliable sign of hip disease during the stage of onset ; the procedure recommended has the decided advantage of being universally applicable, even in the case of very young children. If there should still exist any doubt as to the presence or absence of hip disease, the examination described should be repeated from week to week until one is able to arrive at a definite conclusion.

Second Stage.—The patient now presents more evident symptoms of hip disease, and usually exhibits the attitude of *abduction, eversion, and flexion.* This stage probably corresponds with commencing disease of the articular surfaces and progressive involvement of all the structures of the joint. The attitude described would appear to be the result of muscular action. The original view of Bonnet, that it is caused by distension of the capsule of the joint, is no longer accepted. The most reasonable *explanation of the various attitudes assumed in hip disease* is that given by König ; if the patient walks without crutches, as he is usually able to do at this stage, the attitude of abduction, eversion, and slight flexion enables him to save the limb to the utmost extent ; on the other hand, if he uses a crutch, as he is obliged to do at a more advanced stage of the disease, he no longer uses the limb for support, but draws it upwards and inwards into the position of adduction, inversion, and greater flexion ; similarly, if he is confined to bed he lies on the sound side, and the affected limb sinks by gravity so as to lie over the normal one in the position of adduction, inversion, and flexion. König's explanation accords with the fact that in the exceptional cases which begin with adduction and inversion, we have usually to deal with a more severe type of the disease associated with grave osseous lesions, precisely those cases in which the patient is compelled to adopt the use of crutches or to lie up at an early period. Similarly the transition from the abducted to the adducted position is usually associated with such an aggravation of the symptoms that the patient is no longer able to walk without assistance.

The combination of abduction, eversion, and flexion, although characteristic of the stage of hip disease at present under consideration, is not constant ; the proportion of the different elements not only varies in different cases, but one or other element may be wanting, *e.g.* abduction and eversion may occur without flexion.

The abnormal attitude is at first entirely maintained by the action of muscles, but where it is very prolonged or becomes permanent, the muscles, fasciæ, and ligaments undergo shortening, so that it is continued without muscular effort.

Compensation of Abduction, Eversion, and Flexion ; apparent Lengthening of the Limb.—On first looking at the patient the abnormal attitude may be largely concealed, because the patient usually restores the parallelism of the limbs, by lowering the pelvis on the affected side, and adducting the sound limb ; this obliquity or tilting of the pelvis causes apparent lengthening on the diseased side ; it is best demonstrated by drawing one straight line between the anterior iliac spines and another to meet it from the ensiform cartilage through the umbilicus ; if the pelvis is in its normal or horizontal position, the two lines intersect one another at right angles ; if it is tilted, the angles at the point of intersection are unequal. The flexion of the thigh may be largely compensated for by increasing the forward curve of the lumbar spine (lordosis), and by flexing the leg at the knee. To demonstrate the lordosis the patient should be laid on a flat table ; when the thigh is flexed the lumbar spine is in contact with the table ; when the thigh is extended the spine arches forwards so that one may insert the hand or the closed fist beneath it. In order to estimate the angle of flexion in any individual patient he should lie with the spine pressed flat on the table ; the point is then determined, when, on attempting to extend the thigh, the spine begins to arch ; the angle which the thigh forms with the surface of the table can be readily measured.

There may also be an attempt to compensate for the eversion of the limb by rotating the pelvis forwards on the affected side.

If *the functions of the joint* are tested by the methods already referred to, it will be found that there is more or less complete rigidity, and that both active and passive movements take place at the lumbo-sacral junction instead of at the hip. While the rigidity is usually absolute as regards rotation, it may sometimes be possible with care and gentleness to obtain a little increase of flexion ; for diagnostic purposes most stress should therefore be laid on the presence

or absence of the movement of rotation; for example, if a patient exhibits abduction and flexion, and it is possible to elicit rotation movements, the probability is that he is not suffering from disease in the joint, but from some affection in its neighbourhood.

Swelling, although rarely a prominent feature, should be looked for on the anterior aspect of the joint; when present it may fill up the fold of the groin, and may project forwards the femoral vessels; it is doughy and elastic like the white swelling of other joints, but may at any time liquefy and form a cold abscess; swelling about the trochanter and neck of the bone may be estimated by measuring with calipers and comparison with the sound side; the absence of swelling does not preclude the existence of grave lesions in the interior of the bone. Swelling on the pelvic aspect of the acetabulum may be discovered by rectal examination.

Third Stage.—The patient now presents unmistakable symptoms of hip disease characterised by adduction, inversion, increase of flexion, and apparent or real shortening of the limb. It probably corresponds with well-developed caries of the articular surfaces since pain is now a more prominent feature, and there are usually "startings at night." This stage is less often met with nowadays because the diagnosis and treatment of the disease in its earlier stages are better and more generally known. As far as *the attitude of the patient* is concerned, *the flexion* is usually so pronounced that it can no longer be concealed by lordosis, so that when the patient is recumbent, although the spine is arched forwards, the limb is still flexed both at the hip and at the knee; when the spine is flat on the table, the flexion of the thigh may amount to as much as a right angle. The *adduction* varies very much in degree; when moderate in amount it is compensated for by raising the pelvis on the affected side; this results in *apparent shortening* of the limb; it is of great importance to understand the cause of this shortening, since it can be prevented by treatment.

It is the result of an effort on the part of the patient to restore the normal parallelism of the limbs, the sound limb being abducted to the same extent as the affected limb is adducted. As a result of the obliquity of the pelvis, the patient when erect exhibits a scoliosis of the spine with the dorso-lumbar convexity to the sound side.

When the adduction is very pronounced the patient is unable to restore the normal parallelism of the limbs, and the knee on the affected side may cross the sound limb; there is a deep groove at the junction of the perineum and thigh, the pelvis may be tilted to such an extent that the space between the lower ribs and the iliac crest scarcely admits the fingers, and there is great prominence of the trochanter. In the slighter degree of adduction, which is more often met with, the toes of the affected limb rest on the dorsum of the sound foot, as in a traumatic dislocation on to the dorsum ilii.

The attitude described above is at first the result of muscular action, but when it is prolonged or permanent, the fasciæ, ligaments, tendons, and especially the adductor longus, become shortened, so that it may be impossible to correct it without division of the contracted soft parts.

If the case is left untreated further changes may take place as a result of the pressure of the carious articular surfaces against one another, viz. absorption of the head of the femur, progressive excavation and displacement of the acetabulum, so that the upper end of the femur is drawn gradually upwards and backwards within the socket. Examination will then reveal the existence of a variable amount of *actual shortening*; it will also be found that the trochanter is displaced above Nélaton's line, and that it is nearer the middle line of the body, while above and behind the trochanter there is a prominent hard swelling corresponding to the margin of the enlarged acetabulum.

At this stage there may be a combination of real and of apparent shortening which may amount to several inches. If, as very rarely happens, the original attitude of abduction has been maintained throughout, we may have a combination of apparent lengthening and of actual shortening.

In cases of long standing there may be an additional cause of shortening in the shape of retardation of the growth of all the bones of the limb. Shortening from dislocation will be referred to later.

During the third stage *the other signs and symptoms* become more pronounced: the patient becomes pale and thin; he is usually confined to bed; his sleep is disturbed by "startings" of the limb; the rigidity of the joint and the wasting of the muscles are very striking. Any elevation of the temperature is exceptional; it may rise slightly after a prolonged examination of the limb, or after a railway journey, or from abscess formation. Any marked elevation of the temperature, apart from septic complications, should make one suspect the development of tubercle elsewhere, or of general tuberculosis.

Abscess Formation in Hip Disease.—The formation of abscess is not related to any particular stage of the disease; it may occur before there is any deformity, or it may be deferred until the disease has apparently recovered. The site of abscess and the direction in which it spreads is influenced by its seat of origin and by the anatomical arrangement of the parts concerned. The abscess may be solitary, or there may be several. They appear either in the thigh or in the pelvis, more commonly in the thigh.

The abscesses which form in the thigh usually originate from the cavity of the joint; less frequently they are periarticular; they may present in front of or behind the joint.

(1) *The anterior abscess* in exceptional cases ascends on the deep surface of the psoas muscle into the iliac fossa; it usually appears in the thigh, and comes to the surface on one or other side of the psoas; *the antero-external abscess* appears between the psoas and the great trochanter, a little below the anterior superior iliac spine; from the resistance offered by the fascia lata, it may gravitate down the thigh between the sartorius and tensor fasciæ femoris before perforating the fascia and erupting through the skin; *the antero-internal abscess* emerges at the inner border of the psoas, immediately above the trochanter minor, and bulges in the adductor region; it may perforate the pectineus muscle and point below Poupart's ligament to the inner side of the femoral vessels, or it may point anywhere from the perineum to the middle of the thigh. In connection with these anterior abscesses it occasionally happens that when they have opened and become septic the femoral vessels may be eroded, leading to serious and sometimes fatal hæmorrhage.

(2) *The posterior abscess* develops in the gluteal region, and may make its way to the surface through the gluteus maximus, more often it points at the lower border of this muscle in the region of the great trochanter, occasionally it gravitates down the thigh, or extends upwards towards the posterior iliac spine. Mention must also be made of *the extra-articular abscess in the region of the great trochanter*, which is often quite independent of the joint, and usually ruptures in the neighbourhood of the trochanter; also of *the abscess which originates in the lymphatic glands* along the femoral or external iliac artery, which may lead to the formation of tuberculous ulcers and occasionally to erosion of the femoral vessels.

The abscesses which form within the pelvis originate either in connection with the acetabulum or in relation to the psoas muscle where it passes in front of the joint. Those which are directly connected with disease of the acetabulum may remain localised to the lateral wall of the pelvis, or may spread backwards towards the hollow of the sacrum, or open into the bladder or rectum, or ascend into the iliac fossa and point above Poupart's ligament (usually to the outer side of the iliac vessels), or descend towards the ischio-rectal fossa and perineum and point in the vicinity of the anus, or pass through the great sacro-sciatic notch and point in the buttock. The abscess which develops in relation to the psoas muscle may be shaped like an hour-glass, one sac occupying the iliac fossa, the other filling up the adductor region of the thigh, the two sacs communicating with each other through a narrow neck beneath Poupart's ligament.

It has been well pointed out by Cheyne that *the key to the diagnosis of the point of origin of any pelvic abscess resulting from hip disease*, lies mainly in the examination per rectum; if a swelling is felt on the pelvic aspect of the acetabulum it is probable that the abscess has started there, and one may be able to recognise fluctuation between the iliac swelling and that felt per rectum. If there is no swelling in the region of the acetabulum, the probability is that the abscess has originated in the front of the joint, and has spread upwards into the iliac fossa along the psoas muscle.

The formation of abscess in relation to hip disease would be of comparatively little importance, were it not that by rupturing on the skin surface it results in the *septic complications* which are so disastrous, not only by aggravating the tuberculosis, but also by impairing the general health. The development and progress of abscess, so long as the skin is unbroken, is usually unattended with any fever, or complaint of pain, since unless the patient is carefully examined from time to time the existence of abscess may escape recognition. When the abscess bursts externally septic infection is almost inevitable; this may result in abundant purulent discharge, or where the external opening is insufficient for drainage, in repeated attacks of inflammation attended with fever, burrowing of the pus, and the formation of fresh sinuses, until the condition of the patient may merge into that of hectic fever or chronic septic poisoning, in which he loses ground from day to day, becomes the victim of waxy disease in the viscera, or dies of exhaustion, tuberculous meningitis, or general tuberculosis. We do mean to say that the presence of septic sinuses is incompatible with recovery, but they are a source of danger to which the patient should never be exposed if he comes under treatment with unbroken skin.

Dislocation.—True dislocation is a rare complication, and should never be allowed to take place in a patient who is under observation. It is more likely to occur during the stage of adduction with inversion. It is often ascribed to slight violence, but it is known to have taken place during sleep from the same spasmodic contractions of muscles that give rise to "startings" at night. In the more common dorsal dislocation, adduction and inversion are pronounced, the trochanter projects above and behind Nélaton's line, and the head of the bone may be felt on the dorsum ilii. In the upward or supracotyloid dislocation the head lies below the anterior superior iliac spine, flexion is little marked, eversion may replace inversion, the trochanter is farther from the middle line than on the sound side, the adduction is compensated for by tilting of the pelvis, so that there may

be a combination of actual and apparent shortening.

It is a remarkable fact that when dislocation has occurred there may be less complaint of pain or of "startings" than before, and passive movements may be elicited which up till then were impossible.

Bilateral Hip Disease. Crossed-Leg Deformity.—In the same way that any two or more joints may be simultaneously or successively the seat of tuberculous disease both hip-joints may become affected, each running its course independently of the other. The peculiarity of the bilateral affection is that the patient is necessarily confined to the recumbent posture until the disease has completely recovered, and that his capacity for standing and walking in the future may be seriously impaired, especially if the disease has cured with rigidity or ankylosis in both joints, and still more so if the joints have become fixed in an undesirable attitude; the length and the attitude of the limbs may not be the same; the most striking deformity is where both limbs are adducted so that they cross each other, this being known as the "scissor-leg" or "crossed-leg" deformity, in which the patient, if able to walk at all, does so by alternate forward movements from the knees. Where there is bilateral ankylosis the treatment consists in mobilising one joint by osteotomy or resection, and according to the result obtained, resting satisfied with the ankylosis on the other side, or correcting it by osteotomy or excision. If both hips have become rigid in the abducted position, one must be made movable by osteotomy or excision.

Diagnosis of Hip Disease.—The diagnosis is to be made not only from other affections of the joint itself, but also from morbid conditions in the vicinity of the hip, since in any one of these the patient may be brought for advice on account of pain in the region concerned and a limp in walking. Whenever possible the patient should be stripped; if able to walk, his gait should be observed; he is then examined in the recumbent position, and attention is directed to the comparative length of the limbs, to the attitude of the limbs and pelvis, and to the movements at the hip-joint, especially those of rotation. Whenever there is any doubt as to the diagnosis, both hips should be skiagraphed; the examination above described should be repeated at intervals of a week or ten days until a conclusion is reached. In children there are three non-febrile conditions attended with a limp and with shortening of the limb, which may be mistaken for hip disease, viz. *congenital dislocation, coxa vara, and infantile paralysis*, but in all of these the normal movements are less restricted than in disease of the joint, and the history of onset would be different. Cases of hip-joint disease which have recovered after the occurrence of dislocation may be difficult to recognise from the congenital variety in the absence of a trustworthy history, since the range of movement may be the same in both affections.

In sacro-iliac disease the pelvis may be tilted, and the limb apparently longer on the affected side, but the movements at the hip are retained.

In disease of the great trochanter the resemblance to morbus coxæ is very close indeed; there may be abduction, eversion, impairment of mobility, and swelling in the region of the trochanter, going on to abscess. The movements are less restricted than in disease of the joint, still the diagnosis may not be completed until the swelling about the trochanter is explored by operation, and this should not be delayed otherwise the disease may extend to the joint. The same remarks apply to *disease of the bursæ over the great trochanter*, especially of the bursa beneath the aponeurosis of the gluteus maximus.

In *lesions of the psoas muscle*, or of the *bursa underneath the psoas*, the limb may be flexed and everted, there may be lordosis, the patient may walk with a limp, and there may be no other signs of spinal disease; the diagnosis is made by testing the movements at the hip; in commencing psoas abscess, the movements are only restricted or absent in the direction opposed to the existing attitude, whereas in hip disease they are restricted or absent in all directions, especially abduction and rotation.

Appendicitis in children, when associated with flexion, has been mistaken for hip disease through omitting to test the movements of the joint in other directions.

Disease of the lymphatic glands in the groin may cause flexion and a limp in walking; it should be remembered that tuberculous lymphadenitis may be met with as a complication of hip disease, *i.e.* the two conditions may coexist.

Growth fever and other mild forms of osteomyelitis of the neck of the femur may simulate hip disease, but they are more likely to be attended with pyrexia, there is greater tenderness over the bone, the movements at the joint are less restricted, and there is more decided improvement after resting in bed.

Gummatous epiphysitis is a very rare affection met with in syphilitic children; it is attended with greater swelling and tenderness, and there is less voluntary power over the limb.

Sciatica has been mistaken for hip disease as a result of careless examination; in cases of sciatica flexion at the hip is only restricted when the leg is extended on the thigh.

New growths in the vicinity of the hip, and especially a commencing sarcoma of the upper end of the femur, may be very difficult to differentiate from hip disease, especially where the symptoms develop after a fall, when there is abduction or adduction, and where there is an ill-defined swelling about the trochanter. The movements at the joint are only restricted in certain directions in the case of sarcoma.

Skiagraphs may give definite information. The recognition of egg-shell crackling or of pulsation is only possible in the advanced stages of sarcoma, and is therefore of little value.

The diagnosis from other diseases of the hip-joint is only difficult in exceptional cases.

In *traumatic effusion into the joint* the symptoms follow directly upon the injury, are more definite from the first, and rapidly improve under rest in bed without extension or splints.

Pyogenic affections of the hip, from staphylococcus osteomyelitis of the neck of the femur, from typhoid, from gonorrhœa, may be a source of difficulty especially when they are subacute and do not suppurate; there is usually, however, some pyrexia, there is greater tenderness, and pyogenic lesions may be discovered elsewhere in the body.

Charcot's disease and *arthritis deformans* usually occur under conditions which render any confusion with hip disease most unlikely; the author has, however, recently observed a case of arthritis deformans at the hip in a girl, aged 14, in which no other joints were affected, and in which the resemblance to hip disease was so close that in spite of repeated examination and of successful X-ray photographs, the nature of the case was not cleared up until the joint was explored by operation. In arthritis deformans there is less restriction of the movements at the joint, and if the patient is anæsthetised the rotation movements impart a sensation of roughness or crepitation.

In *hysterical affections of the hip*, while there may be the typical attitude and rigidity of tuberculosis, pain and tenderness are exaggerated, the pains are more diffuse, the tender points vary from time to time, and there is no swelling; when the attention of the patient is distracted it may be possible to elicit movement at the joint or to alter the attitude of the limb; the patient's sleep is rarely disturbed, and the condition may be of long duration without any signs of progress towards destruction of the articular surfaces.

Progress and Terminations. Prognosis.—The prognosis in hip disease is more serious than in tuberculosis of other joints, excepting only those of the spine. It is materially influenced by the stage at which treatment is inaugurated, and by the care and perseverance with which it is carried out. While recovery may take place at any stage, and even in the most unlikely cases, it becomes less likely the longer it has been left untreated. In the initial stage recovery may be attended with a certain range of movement; the capacity of flexing the thigh to a right angle is a great advantage. When the disease has advanced to the stage of destruction of the articular surfaces, the joint will be rigid or ankylosed, and its use to the patient will vary with the attitude of the limb and with the amount of shortening. The average shortening amounts to $1\frac{1}{2}$–2 inches, but it may attain to as much as 6 inches or more. The formation of abscess, especially within the pelvis, is a serious complication in untreated cases. The prognosis is most unfavourable where there are serious lesions of the bones (sequestra) and multiple infected sinuses, especially in adults who are the subjects of tuberculosis elsewhere. Intercurrent disease and unfavourable hygienic surroundings make recovery more difficult. The available statistics show that the mortality from hip disease varies from 10 per cent in cases of "dry" arthritis to 40 per cent in cases with abscess; the risks from abscess, however, have been much reduced in recent years.

The causes of death include meningeal, pulmonary, and general tuberculosis, septic complications, and waxy degeneration. In cases which terminate fatally the average duration of the disease is $1\frac{1}{2}$–3 years. In cases which recover the duration varies indefinitely. Where there are bone lesions and septic sinuses the disease, which may have begun in childhood, may still be active after twenty years or more. It is a safe rule in practice to assume that even mild cases rarely recover in less than two years. The patient or his relatives should be informed of this, and also that they are not to expect recovery without some impairment of function, and that developments may occur which require more prolonged treatment or necessitate operative interference.

Treatment of Hip Disease.—As far as the local disease is concerned we know that a very large proportion of cases recover under conservative treatment, and that the functional results are very much better than those following operative interference, so that unless there are special indications to the contrary conservative measures are always adopted.

Conservative Treatment.—This consists in protecting the limb from injurious strain or pressure and in preventing deformity. Absolute rest is imperative, and the patient must be confined to bed until the pain and tenderness have entirely disappeared and the sleep is undisturbed.

Extension by means of the weight and pulley is of most service where there is pain, spasmodic contractions of muscles, rigidity of the joint, or a deformed attitude of the limb. Its object is to tire out the muscles and prevent the bones pressing injuriously against one another. The weight employed varies from 3–8 lbs. in children to 10–20 lbs. in adolescents and adults. It must be adjusted so as to meet the requirements of each case. If pain returns after having been relieved, the weight should be diminished or stopped for a time. If there is deformity the line of extension should be at first in the direction of the deformity, and should then be gradually altered as the attitude approaches the normal. Flexion yields most readily. In

marked abduction extension should be applied to both limbs. In adduction it may be necessary to apply counter-extension by means of a perineal band. The attitude ultimately desired is one of almost complete extension with slight abduction. It is not necessary to apply the extension plaster to the limb as a whole; abnormal attitudes are more easily corrected if it is limited to the thigh.

The attainment of the desired results requires skill and close personal attention. Extension is too often employed in a routine fashion without regard to the special features of the case. There can be no doubt that the more general use of extension in the treatment of hip disease has been of great service in the prevention of deformity. It is capable of eliminating the common causes of shortening, viz. flexion, adduction, upward tilting of the pelvis, and displacement of the femur within the acetabulum. It cannot prevent the shortening from arrest of growth, but this can be compensated for by a moderate degree of abduction and tilting of the pelvis.

To further promote absolute rest in the attitude of extension and slight abduction additional means may be employed, especially in restless children: *a long splint* may be applied on the sound side, and a heavy *sand-bag* on the affected side; better still is a *double long splint and cross bar.* The long splint on the affected side is made with a hinge opposite the joint, so as to permit of abduction and of varying the amount of it. If there is any tendency to rotation it may be prevented by strips of strong plaster, wrapped round the thigh in a direction opposed to that of the threatened rotation, and fixed to the long splint (or sand-bag) with safety-pins.

Ambulatory Treatment.—When pain, tenderness, and muscular contractions have disappeared, and when any faulty attitude has been corrected (or over-corrected towards abduction), but not till then, the patient is in a condition to benefit from open-air treatment. The joint is still kept at rest, and the affected limb is not to touch the ground. In children who are unable to use crutches we rely upon a double Thomas splint, a Phelp's box, a Bonnet's wire cuirass, or plaster of Paris; by using any one of these the child is converted into a rigid object, capable of being carried about like a portmanteau from one room to another, or into the open air. Personally we have had best results from the double Thomas splint, like that intended for spinal disease, which extends from the occiput to the feet.

When the patient is able to use crutches the affected limb is prevented from touching the ground by fixing a high patten to the sole of the boot on the sound side. The hip-joint is kept rigid in the abducted position by a Thomas or Taylor splint, or by a case of plaster of Paris. The plaster is chiefly used in hospital out-patients who cannot be kept under continual supervision. It extends from the level of the navel to the foot on the affected limb and to the middle of the thigh on the sound limb. The plaster may be strengthened by broad strips of aluminium, and it should be renewed at intervals of from six weeks to three months.

The patient is re-examined from time to time, and the treatment is continued until some time after all symptoms have disappeared, and until that phase of hip disease which is productive of deformity is passed. The duration of treatment will vary from six months to two years. If at any time there is a return of active symptoms the patient must again lie up with extension to the limb. The patient or his relatives must be warned of the tendency to relapse.

Counter-irritation by means of the actual cautery has largely gone out of fashion, but where there is much pain in the persistent hip disease of adults it may give considerable relief. It is applied upon one or other aspect of the great trochanter. When there is *a deformed attitude which does not readily yield to extension,* such as may be met with in cases which do not come under treatment until it has existed for some time, there is considerable advantage in correcting the deformity at once rather than waiting until the disease is cured This may be accomplished under anæsthesia in the majority of cases. If the tendons and fasciæ are so contracted as to prevent this they should be divided. The limb is placed in the attitude desired, and maintained there by the methods already described. When there are other indications for operative interference (see later), the possibility of correcting a deformed attitude may be an additional incentive for recommending it.

Iodoform Injections into the Joint.—This procedure is recommended by Krause and others. The limb is placed in an attitude of slight adduction and inversion; the trocar and cannula, about 2 in. long, is inserted immediately above the trochanter major, and is pushed horizontally inwards at the level of the upper edge of the trochanter until it strikes against bone. This will either be the head or the upper part of the neck of the femur. The limb is then strongly adducted and the trocar is pushed on, always in contact with the femur, until it again strikes bone. This should be the socket. The trocar is withdrawn and the iodoform emulsion is injected through the cannula. The procedure should be repeated at intervals of two or three weeks.

Treatment of Abscess—The early recognition and prompt treatment of abscess before it has enlarged, wandered, or threatened to rupture externally, has greatly reduced the suffering and mortality from hip disease. The methods of treatment are the same as are employed elsewhere, *i.e.* by means of iodoform injections or by

open operation. If the latter is adopted, the opportunity may be taken of clearing out any extra-articular bone lesion at the same time. The wound should heal by first intention, or should be dealt with by the open method from the outset. If the abscess be extensive, or if it have relapsed, or if it be situated within the pelvis, the existence of serious bone disease should be suspected, and the question should be considered whether the joint itself should not be subjected to operative interference, either at the same time as the abscess or at a subsequent period.

Operative Treatment of the Disease in the Joint —Arthrectomy and Excision.—The results of conservative treatment are so superior to those obtained by the operations hitherto practised that we only have recourse to these when there is no prospect of spontaneous recovery. In other words, operation is indicated when the local conditions are such as prevent recovery, or when the general condition of the patient necessitates the removal of the disease. So far as the first of these indications is concerned, the difficulty lies in being able to recognise those cases in which the local conditions are incompatible with recovery at a time when it will be of practical value.

It must be remembered that complete excision of the hip is an operation of considerable severity, as evidenced by the existence of a considerable mortality from shock alone. The occurrence of tuberculous meningitis would appear to be as frequent in cases treated on conservative lines as in those subjected to operation.

Indications for Operative Interference. — 1. When the disease from the outset presents unfavourable features, *e.g.* rapid progress, severe pain, early occurrence of adduction, or when it advances in spite of proper treatment; when pain and starting pains are not relieved by extension; or when, after a long period of quiescence, the disease becomes suddenly active and aggressive.

2. When the disease relapses after apparent cure, and if the recrudescence of the disease is attended with suppuration.

3. When, along with evidence of persistence of the disease, there is a deformed attitude which does not yield to extension; where there is dislocation of the head of the femur, or separation of its upper epiphysis.

4. When there is abscess within the pelvis, or other evidence of disease of the acetabulum.

5. When there are septic sinuses with symptoms of hectic and of waxy degeneration and persistent discharge in spite of proper drainage. These are most unfavourable cases whether an operation is performed or not. Operation may afford the only chance of recovery if it is not too long delayed, but it should not be attempted in hopeless cases.

6. In adults who show no signs of recovery within a year from the onset of the disease. This indication specially refers to "bread-winners," who desire above all things a definite promise as to when they will be able to return to work. Complete removal of the disease is an essential condition for any such promise. In patients over thirty natural recovery is so exceptional that it is unwise to count upon it. In children, on the other hand, there is no urgency for resuming active life; the prospects of spontaneous recovery are great, and an operation may interfere with the subsequent growth of the limb.

Nature of the Operative Interference.—From what has been said it is evident that we are often compelled to operate without knowing the condition of matters within and around the joint. We are, therefore, unable to decide beforehand the nature of the operation which is required. Our object should be to expose the disease wherever situated, and to remove it with the least possible damage to the normal structures. The more "open" the method of operation the better, since more reliable information is obtained by inspection than by palpation. These indications are best fulfilled by Kocher's method, which, by permitting of dislocation of the femur, affords free access to all parts of the joint. If it should be found that the disease in the head of the bone is limited to the surface or to isolated areas, it may be got rid of with the spoon or gouge, thus preserving the head, which is returned into the acetabulum at the close of the operation. This method, which may be described as an *arthrectomy*, should be practised wherever possible. If, on the other hand, there is a gross lesion in the head or neck of the bone, the neck is divided with a chain saw or chisel and the head is removed. The remaining disease having been got rid of, the raw surface of the neck is implanted in the socket, or is made to lie opposite to it. This operation is described as an *excision* of the joint.

In *the anterior operation* (associated with the names of Parker and Hüter) the head of the bone must be removed in order to give access to the acetabulum and posterior segment of the synovial membrane. As this excludes the possibility of arthrectomy, and affords an exposure of the joint inferior to that secured by Kocher's method, its application should be restricted to cases in which the lesion is believed to be confined to the anterior part of the joint.

Kocher's operation is performed as follows:— The patient is turned over on the sound side. An angular incision is made through the skin and fat with its centre at the anterior superior angle of the great trochanter. Its upper limb runs upwards and backwards in the line of the fibres of the gluteus maximus; its lower limb runs downwards and slightly backwards to the posterior margin of the base of the trochanter. The incision is deepened at the upper part of the wound so as to separate the fibres of the

gluteus maximus. At the lower part it divides the tendinous insertion of the same muscle on the outer aspect of the trochanter, exposing the insertion of the gluteus medius. Blood-vessels are ligatured as they are divided. The edges of the divided maximus are held apart. A layer of fat is then divided, exposing the gluteus medius and minimus and pyriformis muscles. The wound is deepened in the interspace between the medius and minimus above and the pyriformis below. By drawing the pyriformis downwards, access is obtained to the posterior surface of the capsule and of the acetabulum. The capsule of the joint is divided along either the upper or the lower border of the pyriformis tendon. The insertions of the gluteus medius and minimus are detached from the trochanter from behind forwards, along with the periosteum and the cortex of the bone. The detachment is to be effected as far forwards as the anterior intertrochanteric line, and to allow of this the thigh should be flexed and rotated outwards. The iliofemoral ligament is detached at the same time. The thigh is then flexed and rotated inwards to allow of the detachment of the insertions of the pyriformis, obturator internus, gemelli, and obturator externus from the inner and posterior surfaces of the trochanter and from the digital fossa. This separation should also include the periosteum and the superficial shell of the bone. In this way the muscles supplied by the superior gluteal nerve (the gluteus medius and minimus) are drawn forwards and upwards towards the tensor fasciæ femoris, which has the same nerve-supply, and which, along with the glutei, is of importance for the future abduction of the thigh, while the remaining muscles, which are mainly supplied by the inferior gluteal nerve, are displaced backwards. When the muscles are drawn apart the posterior surface of the head and neck of the femur and as much as is necessary of the great trochanter are exposed. Branches of the internal circumflex artery running transversely over the capsule at the neck of the femur are secured as they are divided. The diseased synovial membrane may be cut away with scissors from the posterior and upper aspects of the joint. The thigh is now strongly adducted, rotated in, and flexed, the ligamentum teres is divided by cutting on the head of the bone from behind and below; the head is then dislocated backwards, exposing the acetabulum and the anterior segment of the synovial membrane. The acetabulum is cleared of tuberculous tissue, special attention being directed to the removal of caseous or necrotic lesions. If the head of the femur is sound it may be replaced within the acetabulum; if it is diseased it must be removed. Iodoform is rubbed into all the surfaces exposed to the tuberculous infection. The divided ligaments, periosteum, and muscles are in turn sutured with catgut. A drainage-tube is brought out through a special opening behind the trochanter. The limb is fixed in an attitude of marked abduction, so that when the patient ultimately assumes the erect posture he will be obliged to depress the pelvis on the affected side in order to restore the parallelism of the limbs. The apparent lengthening thus induced will compensate, in part at any rate, for the actual shortening. The abducted position is secured by means of the double long splint already described. The drainage-tube is removed the day following the operation. The stitches are removed at the end of a fortnight. In a hospital patient a case of plaster of Paris is then applied for two months; thereafter he begins to walk with crutches.

As regards the result of the operation we are qute satisfied with a close fibrous ankylosis, for this ensures a strong and useful limb, and diminishes the chances of a relapse of the disease.

The anterior operation of Parker and Hüter is performed as follows:—The patient lies on his back. An incision is made through the skin and fat from a point immediately below and external to the anterior superior iliac spine downwards and slightly inwards for about three inches. The fascia lata is divided in the line of the wound. Search is then made for the interspace between the tensor fasciæ femoris and gluteus minimus externally, and the sartorius and rectus internally. The muscles being drawn apart, the capsule of the joint is exposed and freely divided; the head of the femur is then removed after dividing the neck of the bone by means of a broad chisel; the whole interior of the joint is now readily accessible to the finger, and is thoroughly cleared of all tuberculous material; special attention is directed to the neck of the bone and to the acetabulum. The operation is completed by stitching the edges of the divided capsule, the muscles, and fascia lata, and finally the skin. The limb is fixed in an attitude of marked abduction.

If, in the course of either of the operations described an abscess is met with, it should be cleared out at once before opening up fresh layers of tissue. If there occurs a fresh outbreak of tuberculous disease in the scar of the wound, it is probably the result of infection of the tissues exposed during the operation, and it should be promptly dealt with by free excision of the parts involved before it ruptures externally. The formation of abscess at any subsequent period should be dealt with on the usual lines.

Amputation at the hip-joint for tuberculous disease has become one of the rarest operations in surgical practice. It may still be required in cases which have continued to progress in spite of excision, *e.g.* where there is disease of the pelvis or of the shaft of the femur, with septic sinuses, albuminuria, and hectic fever. In such cases the surgeon must be prepared not only to disarticulate at the hip-joint, but also to remove a portion of, or the entire innominate

bone. Such extensive operations will usually be carried out by some form of racket incision, which will permit of the ligature of the main vessels as a preliminary measure.

The Correction of Deformity resulting from Antecedent Disease of the Hip.—The deformities concerned have been already described as accompaniments of hip disease; from neglect or improper treatment they may have been allowed to persist, while the joint disease has recovered. They are associated either with fibrous ankylosis of the joint, or with contracture of the soft parts, or a combination of these conditions. Osseous ankylosis is rare. The contracture of the soft parts specially involves the tendons, fasciæ, and ligaments on the anterior and inner aspects of the joint, and is usually present to such a degree that even if the joint were rendered mobile it would prevent correction of the deformity. It should be borne in mind that the difficulties of correction are so considerable, that it should not be attempted unless the deformity is a serious hindrance to locomotion or a bar to remunerative occupation.

The deformity most often complained of is represented by a combination of shortening, flexion, and adduction.

If flexion is the prominent feature it may be corrected as follows:—The osteotomy is performed through the neck of the femur if it exists (Adam's operation), or through the trochanter if the neck of the bone has disappeared; the contracted soft parts are divided, either by subcutaneous or open tenotomy; the limb is then extended and abducted as described in excision of the joint. In patients whose occupations entail the sitting posture it may not be advisable to correct flexion.

If adduction and inversion are the prominent features the choice lies between a transverse osteotomy below the lesser trochanter (Gant's operation), an oblique osteotomy (from above downwards and inwards) through the great trochanter, or a resection of the joint according to the special features of the case. The adductor muscles, fascia lata, and other contracted soft parts are divided by subcutaneous or open tenotomy. The limb is then forcibly abducted and extended; the extension is maintained by the weight (10 to 20 lbs.) and pulley, the abduction by the double long splint already described. In cases where the deformity is bilateral special efforts should be made to obtain a movable joint, at any rate on one side.

Syphilitic Disease in the Region of the Hip-Joint is scarcely met except in the form of epiphysitis of the upper end of the femur in infants who are the victims of inherited syphilis; it is attended with swelling and tenderness over the neck of the bone, and diminution of the voluntary movements of the limb (pseudo-paralysis); while passive movements may be restricted, this is rarely so pronounced as in tuberculosis.

Pyogenic Diseases

These include a number of diseased conditions resulting from infection of the joint with the common pyogenic organisms, *e.g.* the gonococcus, the typhoid bacillus. While the organisms usually gain access to the tissues of the joint through the blood-stream, a direct infection is occasionally observed from suppuration in the femoral lymphatic glands, or in the bursa under the ilio-psoas. The pyogenic diseases are chiefly met with in childhood and youth.

The clinical features vary with the gravity of the infection; they may be much less striking than is generally supposed. They may assume the form of an acute serous synovitis or hydrops, which recovers, or persists, or relapses after apparent cure. The features are often remarkably latent, especially when the hip affection occurs as a complication of a general illness such as scarlet fever, partly because the patient is confined to bed from the first onset. The hip lesion has been known to escape notice altogether, until at a later period the head of the femur was found lying on the dorsum ilii, or the joint is found to be ankylosed. In the "acute arthritis of infants," resulting from staphylococcus osteomyelitis of the femur or acetabulum, the clinical features may also be comparatively mild, but as a rule they assume a type in which the suppurative element predominates. There is general illness ushered in by a rigor and attended with an irregular remittent temperature; the local features are those of an acute arthritis; there is great suffering and frequently danger to life; the joint is held rigid, there is agony on the least attempt at movement, there are startings at night, the muscles waste rapidly; the limb may retain its normal attitude, more often it becomes flexed and abducted, or adducted; a swelling forms, usually in front of the joint at the upper part of Scarpa's triangle, pushing forwards the femoral artery; the swelling may be obscured by œdema; if untreated the pus escapes from the joint and either gravitates down the limb or, as is more often observed, it bursts through the skin over the joint and discharges by one or more sinuses; the joint becomes disorganised, the head of the bone may be displaced on to the dorsum ilii, or the upper femoral epiphysis may be detached; in the latter case the deformity resembles that which accompanies fracture of the neck of the femur.

As a complication of other diseases, pyogenic affections of the hip-joint are usually met with in relation to pyæmia and septicæmia (including puerperal fever), scarlet fever, typhoid fever, measles, acute rheumatism, and gonorrhœa.

In gonorrhœa the hip-joint is not uncommonly affected; the clinical features may be those of a

serous synovitis with pain, tenderness and fever; the amount of fluid may be so considerable as to cause swelling in front of the joint. The more serious form of the disease is sometimes attended with great suffering and marked flexion, and may result in disorganisation of the joint and ankylosis. Suppuration is extremely rare. It is probable that some of the hip affections met with after childbirth are of gonorrhœal origin.

Progress and Terminations.—In the milder forms of pyogenic disease of the hip, recovery may take place with more or less complete restoration of function; in isolated cases the disturbance of growth at the neck of the femur may result in curvature (coxa vara). In more serious forms there may be persistent sinuses from disease (necrosis) in the femur or acetabulum, there may be fibrous or osseous ankylosis of the joint, permanent deformity from contracture of the soft parts, from separation of the epiphysis, from dislocation, and in children there may be impairment of the growth of the limb. In the gravest forms, especially where several joints or bones are involved, death may take place from general infection or toxinc poisoning.

The occurrence of dislocation has been specially observed in relation to typhoid and other fevers, usually from some slight movement or push, when the limb is flexed and adducted; the dislocation may be the first indication of the existence of the joint lesion; the antecedent condition may be one of serous or purulent synovitis; destruction of bone, either of the head of the femur or of the rim of the acetabulum, is exceptional. If the child has been allowed to recover with dislocation on to the dorsum ilii, he is usually able to walk and run about, but with a limp which becomes more pronounced as he grows up; the features closely resemble those of congenital dislocation, and it may be impossible to distinguish between them unless an accurate history is obtainable. The movements are very free, except in the direction of abduction. The shortening may amount to 2 to 4 inches or more.

The treatment is conducted on general principles, and includes that of any disease of which the hip affection is a complication. The limb is extended by means of the weight and pulley, and kept rigid with the single or double long splint, or by means of sand-bags. If the swelling of the joint persist, and if there is evidence of suppuration, the joint should be explored by an anterior incision inclining downwards and inwards from the anterior superior iliac spine. In children it is remarkable how completely the joint may recover if the pus is promptly evacuated under rigid antiseptic precautions.

If there is *dislocation of the femur* it should be reduced by manipulation with or without preliminary extension; it has been successful in about one-half of the cases in which it has been attempted; with results which have been very satisfactory; preliminary tenotomy of the shortened tendons may be required in some cases. Where reduction by manipulation is impossible, the joint structures should be exposed by one or other of the methods employed in excising the joint; if the obstacle is found to be the filling up of the acetabulum by newly-formed tissue, this must be removed; if the head of the bone has disappeared, the neck, or failing it, the trochanter should be fixed against the acetabulum, thereby diminishing the shortening at the expense of mobility. The author achieved this in one case, after dividing the shortened muscles, by driving a long steel pin through the trochanter into the acetabulum, and fixing the limb in the abducted position; the patient, a girl aged 15, was thereby enabled to bring both heels in contact with the ground and to walk very much better than she had done before. The treatment of gonorrhœal disease of the hip is the same as in other joints.

Acute Osteomyelitis in the Vicinity of the Hip-Joint.—This lesion may originate in the acetabulum or in the upper end of the femur, the latter being the more frequent; in many cases the disease runs its course without suppuration; extensive necrosis is exceptional; separation of the femoral epiphysis is nearly always attended with destructive changes in the joint. The disease usually affects children who are apparently healthy, it begins acutely with shivering, high fever, and severe pain, necessitating confinement to bed from the first onset. A doughy swelling forms in the region of the hip, usually in Scarpa's triangle. If suppuration takes place the features are those of acute abscess, which if left to itself results in the formation of sinuses. Rectal examination is of value in cases which originate in the acetabulum. Treatment is carried out on the same lines as in osteomyelitis elsewhere. The sequelæ which may be met with in neglected cases include contracture, deformity (usually extreme flexion with adduction), ankylosis, shortening of the limb, separation of the upper femoral epiphysis, spontaneous curving of the bone (coxa vara), fracture of the neck of the femur, and dislocation.

Death occurs in a considerable proportion of cases (about 15 per cent).

Acute and Chronic Rheumatism of the Hip-Joint

The joint lesions in acute rheumatism are described under that head. Chronic rheumatism is an ill-defined affection of joints; it is met with at ages varying from 12 to 50 years; it is usually polyarticular, and is chiefly remarkable for the amount of suffering to which it may give rise, and the great disturbance in the functions of the joint which may result from it.

Its claims to be called rheumatic rest upon the facts that it has been observed to follow upon acute articular rheumatism, that it may show exacerbations or relapses attended with pyrexia, and that it is met with in patients who have a family history of acute rheumatism or of inflammations of serous membranes. It is probable, however, that some of the joint lesions described as rheumatic may be of gonorrhœal or other pyogenic origin. While it is regarded as distinct from arthritis deformans, it may merge into that disease, and ultimately present the alterations in the bones which are characteristic of it.

The clinical features of chronic rheumatism of the hip are similar to those observed in other joints; progressive stiffness and deformity are the outstanding features, associated with contracture of the soft parts and fibrous ankylosis of the joint; the patient very often becomes a helpless cripple, and has to spend the rest of his life in bed or on a bath chair.

The prognosis is unfavourable. Treatment is conducted on the same lines as in other joints. This subject is further described in the article "Rheumatism."

Arthritis Deformans—Osteo-arthritis—Malum Coxæ Senile

This disease is comparatively common at the hip, either as a monarticular affection or simultaneously with other joints. While the etiology of the disease in general is obscure, in the hip it frequently develops after an injury.

The changes in the joint are characteristic of the "dry" form of the disease, and are chiefly met with in the cartilages and bones; the synovial changes which are so prominent in the knee are here of little clinical importance. The atrophy and wearing away of the articular surfaces is accompanied by the new formation of cartilage and bone around their margins; the head of the femur may acquire the shape of a helmet, or of a mushroom, or of a limpet shell; from absorption taking place in the neck of the femur, the head may come to be sessile at the base of the neck, and may occupy a level considerably below that of the great trochanter. The acetabulum "wanders" backwards and upwards as in tuberculous disease; it is usually deepened, its floor may project on the pelvic aspect, its margins may form a projecting collar which overhangs the neck of the femur, or grasps the latter so that even in the macerated condition the head is imprisoned in the socket, and the joint is locked. The eburnation of the articular surfaces resembles that seen in other joints. The degenerative changes may extend to the upper part of the shaft of the femur, and result in curving of the shaft and neck, suggesting a resemblance to a point of interrogation, and marked depression of the head of the bone below the level of the great trochanter.

The changes described are necessarily associated with great restriction of the movements of the joint and with striking deformity; the deformity consists in shortening of the limb, usually with eversion, sometimes with flexion and adduction; the trochanter is nearly always displaced upwards and backwards.

The clinical features are those of arthritis deformans in general, and are usually so characteristic that there is little difficulty in the diagnosis. The prominence of sciatic pain in some cases (from the nerve being involved as it passes over the joint) may lead to the disease being regarded as sciatica; the restriction of the movements of abduction and adduction, the presence of cracking and of grating of the articular surfaces, and the aggravation of the pain and stiffness after resting the limb, are helpful in recognising arthritis deformans.

The greatest difficulty is met with in cases where the disease occurs in early life as a monarticular affection, for the resemblance to tuberculosis may be so close that the diagnosis may be impossible until the joint is explored by operation; even skiagraphs may fail to differentiate between them.

The treatment, in addition to that of arthritis deformans in general, consists in maintaining the normal movements of the joint, and in diminishing the pressure on the articular surfaces by the use of sticks or crutches. Shortening of the limb may be compensated for by heightening the sole of the boot. Forcible movements of the joint under an anæsthetic often do more harm than good. Excision of the joint has yielded very satisfactory results; it is indicated where the patient, while healthy otherwise, is neither too old nor too fat, and is unable to walk on account of pain and deformity.

Neuro-Arthropathies

Charcot's disease is met with in individuals, usually men over 30, who are suffering from incipient or well-developed tabes dorsalis. One or both hip-joints may be affected. Sometimes the development of a fluctuating swelling in the upper part of Scarpa's triangle may indicate the presence of hydrops; in many of the recorded cases, however, attention has been first directed to the disease by the occurrence of dislocation from slight violence. The absence of pain and tenderness is always characteristic. When dislocation has occurred the limb is short, the upper end of the femur is freely movable on the dorsum ilii; the rotation arc of the great trochanter may be much reduced as a result of the disappearance of the head of the bone; there may be considerable formation of new bone, giving rise to large tumour-like masses in relation to the capsular ligament and to the muscles surrounding the joint.

When both hips are dislocated the attitude and gait are similar to those observed in bilateral congenital dislocation.

The treatment consists in protecting and supporting the joint; when only one joint is affected advantage may be derived from a Thomas or other splint, along with a patten and crutches; in bilateral cases, from the use of crutches alone.

Hysterical Joint.—The neuro-mimetic or hysterical joint affection is very rare at the hip; most importance attaches to the differential diagnosis, which has been already described under tuberculous disease.

Loose bodies are chiefly met with in association with arthritis deformans and with Charcot's disease; they possess little or no clinical significance.

Diseases of the Bursæ in the Region of the Hip.—The bursæ concerned are—(1) between the ilio-psoas muscle and the anterior aspect of the capsular ligament of the hip, which may communicate with the joint; (2) those in relation to the great trochanter, a superficial one between the skin and the tendon of the gluteus maximus, and a deeper one, often multilocular, between the gluteus maximus and the origin of the vastus externus; (3) over the ischial tuberosity; (4) beneath the attachment of the hamstring muscles to the ischial tuberosity; (5) between the obturator internus and the margin of the small sciatic foramen; this bursa may communicate with another between the tendon of the muscle and the hip-joint. The diseases met with in these bursæ include the so-called traumatic inflammations (trade-bursitis), and those resulting from tuberculosis, pyogenic infection, gonorrhœa, syphilis, gout, rheumatism, etc. (*Vide* article "Bursæ" in vol. ii. p. 40.)

In disease of the bursa beneath the ilio-psoas the limb may be flexed, abducted, and rotated out as in tuberculous disease of the hip or of the psoas muscle; from the first of these the diagnosis is made by testing the movements at the joint, from psoas abscess by the swelling being confined to the region of the hip, viz. at the upper and inner part of Scarpa's triangle.

In disease of the bursa between the gluteus maximus and great trochanter the attitude of the limb may resemble that of hip disease; most difficulty will be found, however, in differentiating between tuberculous disease originating in the bursa and a cold abscess associated with disease in the trochanter itself; the diagnosis is rarely established until the swelling (or any sinus following on its rupture) is explored by operation.

Tumours in the region of the hip are so rare in comparison with other diseases that their existence usually remains unsuspected until their nature is revealed by the progress of the case. The *innocent tumours* include chondromata and osteomata arising from the trochanter or pelvis; the diagnosis is not difficult unless in exceptional cases; for example, *the bursa over an exostosis* may undergo rapid enlargement, and the patient may not be aware of the existence of the exostosis, and applies for advice concerning the bursa, which may be so large that it hides the tumour, and may be mistaken for a chronic abscess or other fluid swelling. The nature of the case may not be recognised until the swelling is incised.

Primary sarcoma may originate in the femur or pelvis; at an early stage the diagnosis from hip disease may be difficult or impossible; it has already been referred to under tuberculosis. The examination of the patient must include that of the rectum and the use of skiagraphs. In the later stages the nature of the disease is evidenced by distension of the cutaneous veins, œdema of the limb, pulsation of the tumour, egg-shell crackling, and the occurrence of spontaneous fracture.

Secondary cancer is by no means uncommon in the upper end of the femur following cancer in the breast, prostate, thyroid, intestine, or liver; it is not recognised as a rule until the occurrence of spontaneous fracture.

As far as *treatment* is concerned it is carried out on the usual lines in the case of the innocent tumours. In primary sarcoma it usually implies disarticulation at the hip by a racket incision, the handle of the racket being so placed as to permit of the ligature of the main vessels at an early stage of the operation. The soft parts are then divided in stages, well above the limits of the growth. There have been successful cases in which part or even the whole of the innominate bone has been removed along with the limb.

Paralysis of the Muscles at the Hip—Paralytic Contracture and Dislocation.—These are chiefly met with in children suffering from spinal or cerebral paralysis. At the first onset the limb is helpless and the joint is flail-like; if recovery takes place to such an extent that the child is able to walk, the attitude and gait may resemble that of congenital dislocation; where the affection is bilateral, the child has been known to walk with the hips acutely flexed so that the chest rests on the knees. The gait may be improved by some form of rigid apparatus which fixes the thigh to the trunk, or by securing an artificial ankylosis at the hip-joint (arthrodesis).

Cases are more often met with in which the child has been confined to bed (or at any rate is unable to walk), and contracture has taken place in the attitude of flexion, adduction, and internal rotation; dislocation on to the dorsum ilii may be superadded. It is of great importance to prevent such deformity by suitable splints and extension; when it has occurred, the question of correcting it will depend on the general condition of the patient and on the functions of the limb, apart from the attitude at the hip. The operative treatment is carried

out on the principles recommended for similar deformities of other origin.

Hippiatria.—Veterinary medicine (from Gr. ἵππος, a horse, and ἰατρός, a physician).

Hippocampus.—A peculiarly-shaped portion of the white matter of the brain (Gr. ἵππος, a horse, and καμπή, a bending or turning); the hippocampus major projects into the floor of the middle horn of the lateral ventricle, and the hippocampus minor into the posterior horn. *See* Physiology, Nervous System (*Localisation of Functions, Taste and Smell*).

Hippocrates. *See* Medicine, History of (*Influence of Greece*).

Hippocratic Facies. *See* Cholera, Epidemic (*Symptoms, algid stage*); Facies; Peritoneum, Acute Peritonitis, General (*Symptoms, Facies Hippocratica*).

Hippocratic Fingers.—The broadening of the terminal phalanges of the fingers, producing "clubbing," along with cyanosis of the digits; the condition is met with in chronic pulmonary disorders (phthisis, bronchiectasis, emphysema) and in congenital heart disease, etc. *See* Fingers (*Appearances in Certain Diseases*); Jaundice (*Prognosis*); Liver, Diseases of (*Hypertrophic Biliary Cirrhosis, Symptoms*).

Hippocratic Succussion. — The splashing sound heard, on auscultation, of the chest when the patient's body is shaken, in cases of hydropneumothorax and pyopneumothorax. *See* Lung, Tuberculosis of (*Physical Signs, Auscultation over large vomicæ*); Pleura, Diseases of (*Acute Pleurisy, Physical Signs, Succussion*); Pleura, Diseases of (*Pneumothorax, Clinical History, Auscultation*).

Hippocratism.—The Hippocratic doctrine of endeavouring to cure disease by the study and imitation of nature's efforts and methods of securing recovery.

Hippol. — The registered trade name of methylene hippuric acid; the drug has been used as a urinary antiseptic, in doses of 15 grains, in cases of cystitis, etc.

Hippuria. — An excess of hippuric acid (*q.v.*) in the urine, as in cases where benzoic acid is being taken.

Hippuric Acid.—Benzamidoacetic acid, formed apparently from benzoic acid taken in the food, linked to glycocoll or amido-acetic acid in the kidneys. *See* Physiology, Excretion (*Urine, Nitrogenous Substances, Hippuric Acid*); Urine, Pathological Changes in (*Sediments, Hippuric Acid*).

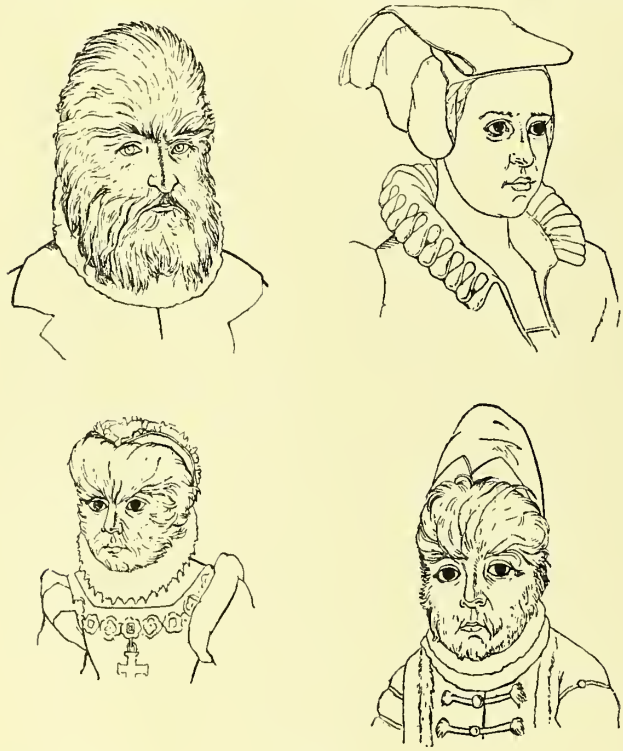
Hippus. — Rhythmical contraction and dilatation of the pupil, as in head-shaking and hysteria. *See* Head-Shaking (*Clinical Features*); Pupil (*Movements and Size*).

Hirsuties. *See also* "Esau"; Pregnancy, Intra-Uterine Diseases (*Skin Affections of Fœtus*); X-Rays (*in Treatment*).

Hirsuties, hypertrichosis or excessive hairiness, is a term having a somewhat wide range of application. The old woman who develops scattered hairs upon the chin, and the old man with bushy eyebrows and a copious growth in the nostrils, external ears, and over the body, are both instances of hirsuties or hypertrichosis of the senile type. The adult man whose body, either in a special and unusual locality or over its whole surface, is provided with hair, and the adult woman whose hairy covering resembles in extent and distribution the male type, are examples, the one of heterotopic, and the other of heterogenic hypertrichosis. Further, at the period of puberty the hair which then normally appears in both sexes may be excessive, and the girl at this time may show the arrangement and development of hair which belong to the boy; again, the appearance of the hair at puberty may be precocious in either sex: these, likewise, are hypertrichoses. There are also hypertrichoses which are due to injuries and diseases of nerves, to trophic disturbances, and to chronic inflammatory states. The nævus which carries hair on its surface (nævus pilosus) has by some writers been regarded as a hypertrichosis, but it is advisable to restrict the use of the term to the cases in which the underlying skin is apparently normal. Finally, the infant at the time of birth, or very soon thereafter, may show a general or a localised excessive growth of hair to which the name of *congenital hypertrichosis* (*universalis, localis*) is correctly given. In the other varieties, also, congenital predisposition may, and doubtless does, play an important part.

If birth occur prematurely the infant will show a sort of physiological hypertrichosis universalis, for the lanugo of fœtal life will still be present. This, however, is not what is meant by general congenital hypertrichosis, which is rather the persistence till birth at the full time and throughout postnatal life of this same lanugo, more or less altered in its physical characters. It is not yet definitely known in what relation excessive hairiness stands to the fœtal lanugo, and it is, therefore, not justifiable to define hypertrichosis as a persistence of the lanugo. Accurate reports are much needed of the condition of the hair at and immediately after birth in the subjects of this trichogenetic anomaly; doubtless this lacuna in our knowledge will ere long be filled; and we shall then know with some certainty whether the lanugo itself becomes the hair of the "hairy infant" or whether it falls off and is replaced by an entirely new growth.

Various names have been given to general congenital hypertrichosis, among which are *polytrichia*, *trichauxis*, *hirsuties adnata*, *dasytes*, *pilosism*, and *hypertrichiasis*. Individuals affected with the anomaly in its most marked form have been called "hairy men," "homines pilosi," "human monkeys," "missing links," and "Esaus." German equivalents are "Haarmenschen," "Waldmenschen," and "Hundemenschen"; and in French the expressions "les hommes velus," "les hommes des bois," and "les hommes-chiens" are met with.



FIGS. 1-4.

Cases of general hypertrichosis congenita are rare. The first recorded case seems to have been that of Esau, who "came out red all over like a hairy garment" (Genesis xxv. 25), or more literally "all of him as a cloak of hair." The meaning of this hairy birth has greatly puzzled the commentators, and Kalisch pointed to it as "a foreboding of the animal violence of Esau's character." In the Middle Ages there was a difference of opinion also as to whether or not Esau's state constituted a monstrosity, and Pohlius, in 1669, wrote a work with the interrogative title, "De Questione an Esau fuerit Monstrum." Among other historical examples was the girl born near Pisa, hairy all over ("totam hirsutam"), whose mother had been gazing at a picture of John the Baptist during her pregnancy (*vide* T. Fienus, *De viribus imaginationis*, p. 224, 1635); and there was the hairy child belonging to the Ursini family, who had bear's claws as well as the hirsute covering. There was the remarkable hairy family ("homines sylvestres,") from the Canary Islands described by U. Aldrovandus (*Monstrorum Historia*, p. 16, 1642); and there was also "Die haarige Familie von Ambras," consisting of a hairy man, his wife normal in the matter of hair, and his hairy son and daughter (Figs. 1-4), described fully by C. T. von Siebold (*Arch. f. Anthrop.*, ix. 253, 1877–8). Another well-known example of hypertrichosis was "Barbara Ursler," who was publicly exhibited in London in 1655, and who is described in Caulfield's *Portraits, Memoirs, and Characters* (vol. ii. p. 168, London, 1794–5), and has been recently considered by Stricker (*Arch. f. path. Anat.*, lxxi. p. 111, 1877). John Crawford, who studied medicine in the University of Edinburgh in the early years of the past century, and who was afterwards envoy to the Court of Ava, brought before the notice of European authors the famous hairy family of Burma (*Journal of Embassy to the Court of Ava*, London, 1834); and many others have since contributed details regarding this family. It consisted of a hairy man married to a normal woman, of his hairy daughter, and of two hairy grandsons, the children of the daughter by a normal man; the dentition of these individuals seems to have been defective (*vide* J. J. Weir, *Nature*, xxxiv. 223, 1886). Reference must also be made to the hairy Mexican woman, Julia Pastrana, described by J. Z. Lawrence (*Lancet*, ii. for 1857, p. 48), H. Beigel (*Arch. f. path. Anat.*, xliv. 418, 1868), F. L. Neugebauer (*Kilka slow o mezkiem owlosieniu u Kobiet*, 1897), and by J. Ranke (*Verhandl. d. München. anthrop. Gesellsch.*, 1–4, 1888); she seems to have had hypertrophy of the maxilla (E. Magitot, *Gaz. méd. de Paris*, 4 s. ii. 609, 1873). In Chowne's case (*Lancet*, i. for 1852, pp. 421, 514; ii. for 1852, p. 51) the hairiness was widespread although hardly universal; the patient, a woman, had a hairless brother and one hairy sister (Wilson, *Lectures on Dermatology*, p. 102, 1878). The girl Teresa Gambardella, described by C. Lombroso (*L'uomo bianco e l'uomo di colore*, p. 155, 1871), resembled Chowne's patient to a certain degree. Then there were the famous Russian "hairy men" or the "Kostroma people" described and discussed by many authorities (E. R. Perrin, *Bull. Soc. d'anthrop. de Paris*, 2 s. viii. 741, 1873; C. Royer, *Ibid.*, p. 718; C. S. Tomes, *Brit. Med. Journ.*, i. for 1874, p. 413; R. Virchow, *Berl. klin. Wchnschr.*, x. 337, 1873; G. T. Jackson, *Med. Record*, New York, xxvii. 568, 1885; and A. Ecker, *Gratulationsschrift*, Braun-

schweig, 1878); these two men (father and son?) had a very remarkable skye-terrier appearance, they were both nearly edentulous, and their nails were soft and thin (J. Parreidt, *Deutsche Monatsschr. f. Zahnhlk.*, iv. H. 2, 1886). Finally, among the well-known instances of hypertrichosis, there was Krao, "the missing link," who was seven years old when she was exhibited by Farini in London in 1883. When seen by A. H. Keane (*Nature*, xxvii. 245, 1882–3) she was of average intelligence, her face and low forehead were covered down to the bushy eyebrows with deep black, lank, and lustreless hair, Mongoloid in type; her whole body was overgrown with a less dense coating of soft black hair; the skin beneath was dark olive brown; the feet were prehensile, and the hands could be bent back at the wrists; and there was slight prognathism. She was said to be the child of Siamese parents (*Nature*, xxvii. 579, 1882–3). Fauvelle (*Bull. de la Soc. d'anthrop. de Paris*, 3 s. ix. 439, 1886), when Krao was about eleven years old, found the second dentition complete, save that the upper canines had not yet been cut.

From the preceding summary of the best known of the recorded cases of congenital hypertrichosis, certain outstanding characters in the *clinical history* and *symptomatology* will have been recognised. Heredity has been very evidently present in several cases, as in the von Ambras Family and the Hairy Family of Burma; family prevalence, also, was noted in several instances. In two cases reported by P. Michelson (*Arch. f. path. Anat.*, c. 66, 1885) these characters were also present: in one, the hairiness affected a man (Joseph Fieber), a native of Silesia, his eldest daughter, his mother, and two brothers; in the other, the father was the subject of hypertrichosis, and so were two of his sons. In both of Michelson's family histories defective dentition was present, and it was sometimes transmitted along with the hirsuties and sometimes apart from it. The sisters Francina and Fytje P., described by Geyl (*Biol. Centralbl.*, viii. 332, 1888-9), were examples of the minor degree of hypertrichosis universalis. Lina Naumann, the hairy girl, seen by L. Fürst (*Arch. f. path. Anat.*, xcvi. 357, 1884), was, however, an exception to the above rule, for she was apparently the only member of her family affected; but she resembled Krao and Julia Pastrana in the possession of normal teeth set on hypertrophied alveolar margins. Marietta S., also, reported by C. Hennig (*Jahrb. f. Kinderhlk.*, xl. 107, 1895), seems to have been a solitary instance of hypertrichosis; but from the description it would appear to have been a case complicated with nævus pilosus.

Details of the state of the hairy infants at birth are sadly lacking. In Geyl's two patients (*loc. cit.*) marked hair on the scalp and long lanugo on the forehead and cheeks were present at birth, but at the age of two and a half years there was a sudden increase in the hair over the limbs and body. In Fürst's patient (*loc. cit.*) the abnormal hairiness of the body was clearly visible within the first week of life, and bushy eyebrows were noticed at birth. In the "homo hirsutus" described by Krebs (*Hosp.-Tid.*, 2 R. v. 609, 1878) the excessive hairiness did not appear until the third month of life. It was usually found that the face and hands were specially hairy, and this gave a very characteristic animal appearance to many of the individuals; but in Pickells' patient (*Edinb. Med. and Surg. Journ.*, lxxvi. 316, 1851) the face and hands were free, while the rest of the body was hairy. In some, the hair was very coarse, but in others it was soft and silky; usually it followed the lines of direction taken by the lanugo in fœtal life. The hypertrichotic condition apparently did not interfere with postnatal existence in any of the recorded cases, and it was not associated with sterility. There was sometimes a correlative variability seen in the dental development, and reference has been made to the alveolar hypertrophy in Julia Pastrana and others; but sometimes there was apparently compensatory defective development of the teeth, as in the Russian "hairy men." It may be noted here that congenital alopecia has also been found associated with dental defects (*vide infra*), and Magitot (*loc. cit.*) has referred to it both in hairless men and in the hairless Chinese dogs. The Ainos of Japan are distinguished from Mongolian and Japanese peoples by a sort of racial hypertrichosis; they also show a marked development of the alveolar border of the superior maxilla with consequent prognathism (Ashmead, *Sei-i-kwai Med. Journ.*, xiv. 183, 1895).

The *pathogenesis* of hirsuties adnata is closely beset with problems. There seems to be something paradoxical in the idea that this excessive production of hair is an arrested development; but on examination it would appear that the theory of an arrest is better supported by facts than any other. The persistence of the lanugo is undoubtedly of the nature of an arrested development, for normally it is shed before or soon after birth. But is hypertrichosis a persistence of the lanugo? In order to answer this question, it would be necessary to have a knowledge of the state of the hair in "hairy infants" during the first hours of life, and more especially of its microscopical characters; this knowledge is not yet in our possession. We do not know whether in these cases a casting of the hair occurs at birth or not. As has been pointed out by P. G. Unna (*Histopathology of the Skin*, N. Walker's Transl., p. 1151, 1896), if the former be the case, and if the embryonic hair follicles, instead of becoming shorter all over the body at this period of life, retained their double length, then, in spite of the abundance of hair,

it is justifiable to speak of hypertrichosis as an arrested development. But if on the trunk and limbs the ordinary casting of the hair had taken place *in utero* and all the hair follicles had shortened, and if, later, these follicles had (as occurs normally on the scalp) again expanded to the original (double) length, and so given rise to another and a very strong growth of hair, then the condition would be that of a true hypertrichosis, analogous to the hypertrichosis of puberty. Unna is of opinion that both these possibilities may occur, and that while for instance the former view holds with regard to the Russian "hairy men," the latter explains such cases as Krao and Julia Pastrana; he prefers to call the former (the simple persistence of the fœtal hair) "trichostasis" or "hair-stagnation," while the latter is true hypertrichosis. It is easy to exaggerate, as I think Unna does, the difficulty of accepting the theory of an arrest of development; congenital ichthyosis also is characterised by excessive growth (of the stratum corneum), and this is probably due to an anomaly of the epitrichium, likewise an arrest of development. If the theory be correct, then in some instances hypertrichosis is truly a monstrosity rather than a disease, while in others it is more correctly a disease; so that after more than two hundred years we might write again as Pohlius did in 1669, "De questione an Esau fuerit monstrum." In a similar unsettled state we must leave the question of the atavistic nature of congenital hypertrichosis.

No treatment has been proposed or indeed thought of for general hypertrichosis; but, for the localised form, electricity and the radium and Röntgen rays have been employed for cosmetic purposes. The localised form, it may be remarked, has usually been confounded with hairy nævus (nævus pilosus); but it ought to be distinguished from it, for in true hypertrichosis the underlying skin ought neither to be pigmented nor abnormally vascular. No doubt most of the cases of "bearded infants" and babies born with "tails" have been instances of hairy nævus affecting the face or sacral region, and the so-called "bathing-drawers" nævus is a well-known variety of cutaneous pigmentation; but true cases of hypertrichosis localis occur, although rarely. For instance, there was A. H. Dodd's case of lumbar hypertrichosis (*Lancet*, ii. for 1887, p. 1063), and there was also Balmanno Squire's (*Brit. Med. Journ.*, i. for 1893, p. 1265), in which a patch of long hair was present on the side of the neck. L. A. Parry (*Lancet*, i. for 1896, p. 1717) recorded a case of lumbar hairiness affecting two sisters. The so-called "lady with the horse mane" was a case of hypertrichosis localised in the dorsal region; in this case there was a defect in the vertebral column (spina bifida occulta) underlying the hair. This association of lumbar hypertrichosis with spina bifida occulta has been noted by several observers in other cases, *e.g.* by W. Stricker (*Arch. f. path. Anat.*, lxxiii. 624, 1878), by F. von Recklinghausen (*Ibid.*, cv. pp. 243, 373, 1886), by C. Brunner (*Ibid.*, cvii. 494, 1887), by G. Joachimsthal (*Ibid.*, cxxxi. 488, 1893), and by others. In some of these instances there was a further complication which came on in later life, namely, perforating ulcer of the foot. Some of the cases reported as infants with tails were no doubt instances of lumbar hypertrichosis. Bland Sutton (*Lancet*, ii. for 1887, p. 4) wrote suggestively on this subject, as did also Emil Kruska (*Dissert.*, Jena, 1890).

Hirudo.—There are two official species of leech, the green (*Sanguisuga officinalis*) and the speckled (*S. medicinalis*), and both are still occasionally used for the purpose of local blood-letting. The bleeding from the triradiate bite given by the leech may sometimes be difficult to check, requiring the application of pressure or of a hæmostatic. Leeches are now seldom applied to canals, such as the rectum, or to internal organs, such as the cervix uteri. The *Hirudo Australis* (Australian Leech) is described in the Indian and Colonial Addendum to the B.P. of 1898.

Histidin. *See* Physiology, Protoplasm (*Proteids, Diamido Acids*).

Histogenesis.—The formation of the tissues (Gr. ἱστός, a web or tissue, and γένεσις, an origin).

Histohæmatins.—Pigments, allied to hæmoglobin, found in the tissues, such as myohæmatin. *See* Pigments of the Body and Excreta (*Hæmoglobin and its Derivatives*).

Histology.—The study or science of the tissues, their characters and development.

Histon.—A proteid, sometimes found in the urine. *See* Physiology (*Protoplasm, Conjugated Proteids*); Urine, Pathological Changes in (*Proteids in*).

History, Medical. *See* Medicine, History of.

Histosan.—A combination of guaiacol and an albumin, which has been used in the treatment of the different forms of tuberculosis (of the lungs, bones, peritoneum, testis, etc.); it has been given in the form of a powder in doses of from 4 to 8 grains.

Histrionic Mania.—Insanity characterised by the imitation of the movements of a player; dramatism.

Hives.—A name given popularly to various skin diseases of children, including the "red gum" (Strophulus), chicken-pox, and nettlerash; it is also sometimes applied to enteritis (*Bowel-*

Hives), and to croup; its derivation from the verb *to heave* is possible, but is with difficulty brought into consonance with etymology.

Hoarseness. *See* Larynx, Chronic Infective Diseases of (*Laryngeal Phthisis, Diagnosis*; *Malignant Disease of Larynx, Symptoms*); Lung, Tuberculosis of (*Lung Symptoms, Hoarseness*); Mediastinum (*Growths, Symptoms*).

Hobnailed Liver. *See* Liver, Diseases of (*Portal Cirrhosis, Synonyms*).

Hodara's Disease.—A variety of Trichorrhexis Nodosa.

Hodge Pessary. *See* Uterus, Displacements of (*Retroversion and Retroflexion, Treatment*).

Hodgkin's Disease. *See* Lymphatic System, Physiology and Pathology (*Lymphadenoma*); Thymus Gland (*Diseases, Enlargement and Persistence*).

Hoffa's Disease.—Fibrous, pseudo-inflammatory hyperplasia of the adipose tissue under the ligamentum patellæ. *See* Sinding-Larsen's article in the *Norsk Mag. f. Laegevidensk.*, 5 R. vol. iii. p. 248, 1905.

Hoffmann's Anodyne.—*Spiritus Ætheris Compositus*; consists of alcohol, ether, and the heavy oil of wine. The last-named is a complex ethereal compound made by mixing sulphuric acid and alcohol. *Dose*—60-90 m.; 20-40 m. if repeated. It is an excellent carminative, and is serviceable in the treatment of hiccough, flatulence, palpitation from indigestion, tobacco heart, and angina pectoris during the attack. Too rapid volatilisation of the ether gives rise to difficulty in swallowing the drug, and it should therefore be given in capsule or in iced water. It probably owes its action chiefly to the ether contained and not to the heavy oil of wine, as has been generally supposed.

Holland. *See* Balneology (*Holland and Belgium*).

Hollands. *See* Alcohol (*Spirits, Gin or Hollands*).

Holmgren's Test. *See* Colour Vision (*Methods of Testing by Coloured Wools*).

Holoacrania.—That variety of anencephalus or acrania in which there is complete absence of the cranial vault and cerebral tissues. *See* Teratology.

Holoblastic Segmentation.—That form of segmentation in which the ovum (*e.g.* the mammalian ovum) in its entirety is converted into a mass of cells and forms the morula or mulberry mass. *See* Embryology.

Holocaine Hydrochloride.—A substance allied to phenacetin; occurring as small colourless crystals, soluble 1 in 55 of water. It has been used as a substitute for cocaine for operations on the eye. The advantages claimed for it are that it does not dilate the pupil, does not affect intra-ocular tension, does not roughen the corneal epithelium, and has no vascular effects. It is usually employed as a 1 per cent solution.

Hologastroschisis.—The teratological type in which there is defect of the anterior abdominal wall in its whole extent from sternum to symphysis pubis.

Holorhachischisis.—The teratological type in which the whole spinal canal from end to end is open, every one of the vertebral arches being imperfectly developed; it stands in opposition to merorhachischisis, in which the spinal canal is open in one region only. *See* Teratology.

Homalomyia scalaris.—One of the privy flies whose larvæ may be found in the stools in gastro-intestinal myiasis.

Homatropine.—An alkaloid, having a mydriatic effect, obtained from atropine. Homatropine ($C_{16}H_{21}NO_3$), in the form of the hydrobromide ($C_{16}H_{21}NO_3HBr$), may be given in doses of $\frac{1}{80}$ to $\frac{1}{20}$ grain, or applied locally (*e.g.* in eye practice) as the *Lamellæ* (discs) *Homatropinæ*, each of which contains $\frac{1}{100}$ grain of the hydrobromide. *See* Alkaloids; Belladonna; etc.

Homburg. *See* Balneology (*Muriated Waters, Germany*); Mineral Waters (*Muriated Saline*).

Homicide.—The killing of a human being by another human being, classified as justifiable, excusable, and felonious or culpable. *See* Insanity, Nature and Symptoms (*Homicidal Melancholia*); Medicine, Forensic (*Wounds, Homicidal*); Senile Insanity (*Psychoses of Senility, Insane Acts*).

Hominy.—A preparation of maize, undeprived of its proteid, but having only $\frac{1}{2}$ per cent of fat. *See* Diet (*Vegetable Foods, Cereals*).

Homo-.—In compound words *homo-* (Gr. ὁμός, same) has the meaning of *similar*, and is opposed to *hetero-*.

Homo Caudatus.

Not Teratology only but Anthropology and Ethnology are all concerned in the discussions which have taken place regarding the occurrence of "tailed infants"; *homo caudatus* or *uro-anthropos* (Taruffi) has always excited curiosity and aroused controversy. In Mythology, also, the records of tailed individuals and races are not wanting.

A great deal has been written and more has been rumoured about the existence of a tailed race of human beings. From the earliest times of Herodotus and Pliny and Ctesias, up to the most recent reports of modern African explorers, the credulous public has been asked to believe in the presence somewhere on the earth's surface of men with tails. Rumour has been busy with reports as to the geographical location of this tailed race: now, on an old map, we find it in Tierra del Fuego, the abrupt and laconic legend being "homines caudati hic"; again, it is in Polynesia (in New Britain), and the infants that are born without the caudal appendage are, so we are told, summarily killed; again, it is near the sources of the Amazon ("cependant je laisse à chacun ajouter la foi qu'il voudra à ces faits," adds a critical reporter), or in Trebizond in Asiatic Turkey among the descendants of Constantine the Great ("d'ailleurs la puissance de Dieu est infinie; il fait tout ce qu'il lui plaît, et crée, quand il le veut, des objets inconnus aux hommes"), or in Turkestan ("stinkendes Ungeziefer mit Schwänzen"), or in India in Rajputana ("the long-tailed Ranas of Saurashtra"), or in China, or in Japan, or in Formosa, or in the Malay peninsula, and the islands of Borneo, Sumatra, and the Philippines; but in Africa more perhaps than in any other part of the world have there been reiterated "travellers' tales about tails" (in Marocco, among the Niam-Niams of Central Africa, in Loango, in Bornu, etc.). Even in Europe, more especially in the west and south, there are legends, usually of considerable antiquity, of the occurrence of tailed peoples. Scotland and England are not without their records; and the people of Rochester ("illa gens incredula") seem to have been most unfortunate, for by offending first St. Augustine (*Golden Legend*, iii. 201, Temple Series) and later Thomas à Becket, their children were born with tails, as a mark, so the chroniclers say, of God's displeasure. The reader who may desire to know more concerning these quaint beliefs of the past will find what he wants in Bartels' classical papers (*Arch. f. Anthrop.*, xiii. 1, 1881; xv. 45, 1884) and in the many references which they contain. Some articles which have appeared in recent years and which may also be consulted are by Tirant (*Bull. Soc. d'Anthrop. de Lyon*, i. 158, 1881-82; ii. 157, 1883), J. Wilson (*Ztschr. f. Ethnol.*, xii. 74, 1880), T. H. Parke (*My Personal Experiences in Tropical Africa*, p. 397, 1891), P. D'Enjoy (*Anthropologie*, vii. 531, 1896), and Zaborowski (*Bull. Soc. d'Anthrop. de Paris*, 4 s. viii. 28, 1897).

Some parts of the evidence regarding the tailed races have been hearsay, and, therefore, outside the province of scientific inquiry; and other parts of it have been founded upon observations upon tailed individuals made at such a distance as to leave the possibility that the caudal appendages were of the nature of attached ornaments and were not structurally united to the body. The tailed peoples have always shown a tendency to recede as the curious traveller or anthropologist advanced towards them, and when he reached his furthest point it was to find that the "homines caudati" were still three or four days' journey further on. There is no really reliable evidence of a tailed race of human beings, although, as we shall see immediately, there are not a few records of individuals with tails or with caudal appendages resembling tails. "Homines caudati hic" cannot yet be written over any part of the world's surface; but it is more than likely that in every country a tailed baby now and again makes its appearance, to cause loud wonder among the vulgar and suppressed scientific excitement among the learned.

Let us look now at the individual or sporadic cases of caudal appendage which have been put on record at various times and in various places. Some of the older records contain in them a suggestion of the mythical, although the illustrations that accompany them are apparently most convincing. As a type of such ancient and dubious cases I may cite that reported by Krahe (*Phil. Trans.*, xiv. 599, Oxford, 1684). It is entitled "The Description of a Monstrous Child, born Friday, the 29th of February 1684, at a village called Heisagger, distant about four English miles from Hattersleben, a Town in South Jutland, under the King of Denmark's Dominion, communicated by Mr. Christopher Krahe, a member of the Ecclesiastical Consistory and Provost of all the Churches belonging to the said Diocess." The record, then, is most circumstantial, and the reader who doubts may find Hattersleben (or Hadersleben) on the map, in South Jutland, just as is affirmed. The mother of the child was wife of a soldier; the infant's legs looked as if they had been hacked, and there were swellings on them like bullets; and a maternal impression is therefore indicated. "The face did look pretty old"; and at the forehead there were excrescences, "as if it were artificial laces." "With the left eye it did look fiercely, keeping that other close." There were also malformations of the back of the head, of the left arm, and of the toes. The description of the tail (which is shown almost reaching the ground in the figure) is disappointingly short: "the Tail, which was strangely grown out of the back-part, was a quarter of a Scalandish Ell long." The mother had two other sons, alive and well; "but this Monster, after it had cryed out 2 or 3 times, died presently." The tail, long and thin, is represented as springing from between the buttocks just above the anus. It is difficult to know what to make of this record; but the most probable explanation is that an infant deformed in a way resembling the description given was actually born and died where and when it is stated; and that the artist considerably lengthened the tail, and tried to

give some touches of artistic verisimilitude to the lower limbs in accordance with the father's military experiences. A suspicion of the same æsthetic amplification attaches to the illustration which accompanies J. S. Elshott's description of a "Puella monstrosa," who had an umbilical hernia, a left arm ending in a point, deformed digits, and two rat-tail-like appendages, one attached to the right thigh and the other over the tip of the coccyx. By a curious coincidence, the father of this infant was also a soldier (*Miscel. Acad. nat. curios.*, iv.–v., Append. p. 80, 1676). I need cite no others of these seventeenth century cases, for the two already mentioned will serve as types. It is to be regretted that the shadow of doubt rests upon them, for if they were free from it they would stand out prominently as the best instances that we possess of truly tail-like tails in the human subject. The shadow, however, does rest upon them.

Before I proceed to describe some modern examples of tailed infants, it may be well worth while to narrow down the whole subject by excluding the "false tails." In the first place, it is necessary to set aside sacro-coccygeal neoplasms of all kinds. I recently saw (with Mr. Stiles) a female infant with a large tumour attached to the post-anal region which, when examined by the Röntgen rays, was found to contain a spinal column and ribs; obviously this was not a tail but an attached twin or parasitic fœtus. Teratomata, teratoid growths, and dermoid cysts may also grow in this region; Thirk's case (*Oesterr. med. Wchnschr.*, N. 36, 1121, 1847) and Jacob's (*Dublin Hosp. Rep.*, iv. 571, 1827) probably both belong to this group. Lipomata, also, of the sacral region are not tails, although when they become pendulous, as in one of Bartels' cases (*Deutsche Ztschr. f. Chir.*, xx. 100, 1884) and in Fieux' case (*Rev. mens. de gynéc. d'obst. et paed. de Bordeaux*, iv. 456, 1902), they may closely simulate them. All caudal appendages composed of fat are not, however, of necessity lipomata, for Virchow found in one of them (Greve's specimen) some traces of the notochord (*Arch. f. path. Anat.*, lxxii. 129, 1878; lxxix. 176, 1880). Again, the sac of a spina bifida of the sacral region must not be mistaken for a tail (Fig. 2); at the same time a caudal appendage and a spina bifida sac may be associated, as we shall see immediately. Neither can we regard the hairs which sometimes arise over a spina bifida occulta, and grow to some length, as a true tail. These various structures may all become caudiform, but they are to be separated off from human tails.

I have personally been able to examine several cases of "tails" and "pseudo-tails." In one of these there was an open lumbar spina bifida, and below it a thick caudiform growth with a little button-like projection at its lowest point; it seemed to be no more than a lipoma. In another case there was a sacral spina bifida sac; and in another there was a conical projection which seemed to contain the lower end of the spinal column, and from the tip of which an amniotic band took origin. The latter of these two cases has some claim to be regarded as a tail. In another case, which I saw in January 1902, there was a curious combination of spina bifida and tail: the child, a male, was fifteen months old when brought under my notice by Dr. T. J. Thyne; it was born six months after the death (from typhoid fever) of the father, and the mother had one other child, a female, nine years of age, and normally formed; the infant had a sacral spina bifida sac, covered with skin, and measuring 6 inches in circumference, and from the apex of this there projected a tail-like appendage. The "tail" was 1 inch in length, and was covered with normal skin; it was quite soft; it consisted of two parts, a proximal, resembling the first phalanx of the little finger, and a much thinner distal part, more tail-like. Unfortunately, a photograph of this caudal appendage was not obtained. It had resemblances to that described some time ago by Edmund Owen (*Trans. Path. Soc. Lond.*, xxxix. 425, 1888), who compared it to a "fat little finger." In yet another case which came under my notice there was a "tail." This was a fœtus, suffering from an exuberant overgrowth of cartilage in various parts of the skeleton (*chondrodystrophia fœtalis hyperplastica*), and the coccyx, participating in this change, grew into a marked "tail."

What may be called "true tails" may be subdivided into those which contain bone or cartilage and those which do not (soft tails), or into those which are attached or adherent ("die angewachsenen Schwänze," Bartels) and those which are free ("die freien Schwänze," Bartels). Of tails containing bone or cartilage or rudiments of vertebræ there are very few specimens known. T. Bartholin (*Hist. anat. et med. rar.*, cent. vi., hist. 44, p. 268, 1681) and M. F. Lochner (*Miscel. Acad. nat. cur.*, Dec. ii., Ann. vii. p. 230, 1689) have recorded cases in which it is stated that more than five coccygeal vertebræ were present in the tail; but it is a pity that the details are not more convincingly stated, and that Lochner writes "in a merry vein," as Jacob (*loc. cit.*) puts it. No modern instance has been reported of a human tail containing more than the usual number of coccygeal vertebræ, namely, three to five, and even of them there are very few (Voltaire, *Dict. philos.*, xi. 211, 1832; Ornstein, *Ztschr. f. Ethnol.*, xi. 303, 1879; C. Hennig and A. Rauber, *Arch. f. path. Anat.*, cv. 83, 1886). Of specimens containing cartilage or rudiments of the notochord I may refer to those of J. Wilson (*Ztschr. f. Ethnol.*, xii. 74, 1880), of S. Blancard (*Collectanea medico-physica*, Amsterdam, 1680–88), of Fleischmann (1841) and Gerlach (*Morphol. Jahrb.*, vi. 106, 1880), and of J. H. F. Kohlbrugge (*Natuurk. Tijdschr. v. Nederl. Indië*,

lvii. 163, 1898). As an example of this group of tails, I may cite the case described by Hennig and Rauber (*loc. cit., supra*).

The mother of the tailed infant seen by Hennig was a I-para; she was a dairymaid, and had got a fright at the parturition of a cow. Her child, a female, presented by the breech, and there was very little liquor amnii; it was only 29 cms. in length, the lower limbs being short. The upper part of the body was well formed, and the number of vertebræ above the sacrum was normal. The lower limbs showed several malformations (peromely), and there was atresia ani vestibularis and absence of the labia majora. There was a post-anal dimple (*foveola coccygea*) 1 inch above the base of the tail; it corresponded to the apex of the sacrum, and it was surrounded by a ring of hairs. The sacral vertebræ were only four in number. There was a groove from the post-anal dimple to the root of the tail. The tail itself was a little over an inch in length (2·7 cms.), was somewhat triangular in form, was covered with normal skin, had some lanugo hairs at the tip, and possessed a hard core. The core consisted of cartilage, made up of a proximal and a distal part, apparently representing five vertebræ. The tail was somewhat flattened at the tip, and contained, in addition to the cartilage, some muscular fibres (levator caudæ or extensor coccygis, etc.). On more minute examination the two segments of the tail were found each to consist of a small cylinder of bone (diaphysis), with two extremities (epiphyses) of hyaline cartilage; they may be regarded either as vertebræ arrested in development, or as a parasitic appendage, but the authors regard the former as the more probable conclusion. They were attached by a fibrous band to the sacrum.

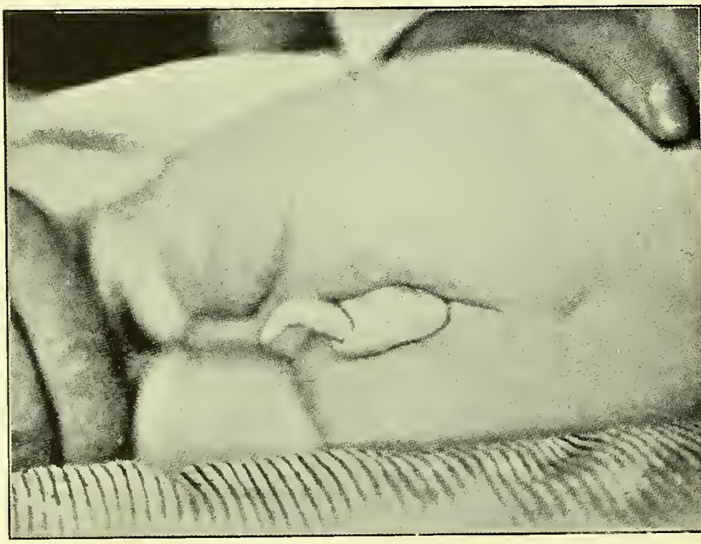


Fig. 1.—Harrison's Patient with Soft Tail.

Of soft tails several specimens have been reported. They may be attached or free: of the former I may mention Labourdette's case (*Journ. gén. de méd., chir., et pharm.*, xxxii. 375, 1808), and those of Bartels (*Arch. f. Anthrop.*, xiii. 1, 411, 1881) and of H. W. Freund (*Arch. f. path. Anat.*, civ. 531, 1886); and of the latter, those of Oskar Schæffer (*Arch. f. Anthrop.*, xx. 189, 1891–92), of Pyatnitski (*Diss. inaug.*, St. Petersb., 1892), of K. N. Vinogradoff (*Vrach*, xv. 901, 1894), of W. Scheboldaeff (*Zemsk. Vrach*, vi. 2, 28, 1893), of R. G. Harrison (*Johns Hopkins Hosp. Bull.*, xii. 96, 1901), and of E. Hagenbach (*Arch. f. klin. Chir.*, lxvi. 426, 1902). The attached tail usually consists of a triangular elevation of the skin covering the lower end of the sacrum; it passes to one side of the anus and may become continuous with the raphé in front of it. Harrison's example of a free soft tail is a typical one, and calls for a brief description.

In the case exhibited in 1900 by Watson (*Johns Hopkins Hosp. Bull.*, xi. 114, 1900) and examined by Harrison (*Ibid.*, xii. 96, 1901), the tail was attached to a male infant, who showed no other anomaly save shortening of the toes of one foot (two phalanges in each). The tail (Fig. 1), which was $1\frac{3}{4}$ inch long at three weeks and $2\frac{1}{4}$ inches at three months, arose about $\frac{1}{2}$ inch below the coccyx. Above its root was a groove leading to a well-marked post-anal dimple (*foveola coccygea*). It was covered with normal skin, had a firm but not a hard consistence, and it had three recognisable segments. The basal segment was short and was evident only on the ventral side; the middle part was an inch in length, and was clearly separated by a constriction from the end segment; the last-named portion was curved to the right and ventrally, and ended in a rounded blunt extremity. There were numerous hairs, especially near the tip. It is very noteworthy that the tail was slightly movable: when at rest it lay extended in the middle line, but, when the child was irritated, it was contracted and the terminal segment was drawn in and flexed to the left side. After the tail had been amputated it was carefully examined. The skin was normal save on the ventral aspect, where there was some thickening of the epidermis. The sebaceous and sudoriparous glands were normal, and the hairs were obliquely inserted pointing towards the tip. The bulk of the tail was made up of areolar tissue containing much fat. There was no trace of notochord or spinal cord; but there were

some bundles of voluntary muscular fibres which took origin from the subcutaneous tissue near the proximal end of the middle segment, and were inserted into the skin of the terminal segment mostly on its left side. No muscles passed from the trunk to the tail. There were several arteries and some small veins, also a number of small nerve trunks.

Before I refer to the mode of production of tails I may take note of the fact that such structures are usually found in fœtuses which show other malformations. Schæffer (*loc. cit.*) has made a careful analysis of the associated malformations: among them were atresia ani, exomphalos, exstrophy of the bladder, asymmetrical development of the whole body, defective limbs, anomalies of the genital organs (*e.g.* hypospadias), amniotic bands, anencephalus, hypertrichosis, polymastia, and club-feet. A tail has also been found in double terata (Hohlfeld, *Diss. inaug.*, Würzburg, 1861; Becker, *Diss. inaug.*, Göttingen, 1889) and in several instances of sympodia. Its association with fusion of the lower limbs (sympodia) is particularly interesting: when it occurs it would seem that in some instances (*e.g.* Ruge's case, *Arch. f. path. Anat.*, cxxix. 381, 1892) the "tail" is due to a dislocation of the lower end of the spine backwards and to one side (as in one of my cases, Fig. 2); while in others it is simply a soft caudiform structure which is met with ("penis-like" in W. Maclaren's specimen, *Edinb. Med. Journ.*, xviii. 658, 1873; xix. 590, 1874; "soft, vascular, spongy, and obtuse" in J. Hofer's *Acta Helvet.*, iii. 366, 1758; like a pig's tail in D. Superville's, *Phil. Trans.*, xli. 302, London, 1744; and with its tip of livid colour and divided into two lobules in B. W. Huesker's case, *Diss. inaug.*, Gryphiæ, 1841).

It is necessary, now, to consider very briefly what explanation Teratology has to offer for the occurrence of such true "tails" as have been described. Embryology supplies part of the answer, and the general principles of Teratogenesis when applied to this part of ontogenesis complete the solution of the problem. There is a stage in the development of the human embryo when it possesses a real tail; it is best marked in embryos of about 14 mms. in length (fifth week); and it may be well seen in Figs. 12, 13, and 14, and especially well in Fig. 17 (*see* article on Embryology, Human, vol. iii. 87-99). Harrison's description of the tail in the human embryo (founded on the examination of Mall's specimens) is good, and I quote from it here. There are seven coccygeal vertebræ in embryos 14 mms. long, and from the third of these onwards is the "tail," *i.e.* the part which projects free from the trunk. Half of the length of the projection is occupied by the vertebræ, while the distal part contains no vertebræ; the former part may be called the coccygeal projection (*Steisshöcker*), and the latter the "caudal filament." The caudal filament is bent towards the dorsum and ends in a slight knob; the medullary cord (spinal cord) runs to the tip of it and ends in a vesicular swelling; the notochord and the terminal branches of the aorta (middle sacral) and inferior vena cava also pass

Fig. 2.—External Appearances, posterior view, of Uromelic (Sympodial) Fœtus with Caudal Projection. Specimen No. 250.

into it, but not to the same distance as the medullary cord; and there is a mesenchymatous network in it with a thickened area, perhaps representing the post-anal gut of earlier embryonic life. At the level of the thirty-second vertebra (just above the base of the tail) the medullary cord suddenly becomes constricted to form the filum terminale; there are few or no neuroblasts beyond this point; and the walls are composed of columnar epithelial cells. As

embryonic life goes on the caudal filament atrophies; the part containing vertebræ (coccygeal projection or *Steisshöcker*) remains for a time as a prominence, but gradually disappears by the growth and alterations in surrounding parts and by the reduction (by fusion) of the seven or six coccygeal vertebræ to five, four, or three. It begins, now, to be clear that these human "tails" are the result of arrested development. The soft, boneless tails are due to persistence of the caudal filament (His), and the rarer cartilaginous or osseous tails are due to the arrest of development in the later stage, when only the *Steisshöcker* is left. We are not in a position to explain completely how the arrest is brought about, or how the development of neighbouring parts affects it or is affected by it; but we may surmise, with Schæffer (*loc. cit.*) and others, that either amniotic compression or amniotic adhesions or both have something to do with it. It is interesting to note that several of the associated malformations (*e.g.* sympodia) are ascribed to amniotic narrowness, and it is quite possible that the caudal fold or part of the amnion may remain attached to the caudal filament or *Steisshöcker* and prevent its normal atrophy or its embedment in the surrounding structures. Human "tails," therefore, would seem to be due to the persistence of a state which is transitorily present in the human embryo itself; no evidence can be extracted from them to support the view that they are atavistic revivals of the animal tail of a far-back ancestor. Whatever may be its origin, the human "tail" had best be removed by the surgeon's knife to relieve the patient from its "discomfort and reproach."

Homœopathy. *See* Medicine, History of (*Nineteenth Century*).

Homologous.—In Pathology a homologous growth is one having the same structure as that of the normal tissue of the part (*e.g.* a myoma of the uterus); in Obstetrics, homologous twins are those born within the same chorion (uniovular or monochorionic), and showing usually a great resemblance to each other; the term is opposed to *heterologous.*

Homonataloin.—A lower homologue of nataloin and found along with it in Natal aloes.

Homonymous.—"Applied to the two images of one object seen in looking at a point nearer than the object, when the right image is that seen by the right eye and the left by the left; opposed to *heteronymous*" (*Murray, N.E.D.*). *See* Retina and Optic Nerve (*Physiology, Homonymous Hemianopia*).

Homopterocarpin. — A substance ($C_{12}H_{12}O_3$) found in red sandalwood (*Pterocarpus santalinus*), along with pterocarpin ($C_{10}H_8O_3$) and santalin ($C_{16}H_{14}O_3$).

Homoquinine. — An alkaloid ($C_{20}H_{24}N_2O_2$) which is a compound of cupreine ($C_{19}H_{22}N_2O_2$) and quinine ($C_{20}H_{24}N_2O_2,3H_2O$); and is also known as *ultra quinine.*

Homotype.—One organ or part is said to be homotypal or homotypic of another when it is of the same structure; a homologue, especially a lateral or serial homologue.

Homunculus.—A mannikin or dwarf. *See* Dwarfism.

Honey.—*Mel Depuratum*: clarified honey. *Preparation*—Oxymel, a mixture of honey, acetic acid, and water. *Dose*—1-2ʒ. It is also contained in Confectio Piperis, Oxymel Scillæ, and Mel Boracis. Honey mixed with water is a good basis for gargles. It relieves dryness of the mouth and fauces, facilitates swallowing, and lessens congestion of the throat by increasing the secretion of mucus. It is frequently given as an ingredient of expectorant mixtures. It has a slightly laxative action, and may be taken by children for this purpose.

Hook.—The term "hook" has been given to a number of different instruments used in Surgery and Midwifery: for instance, various hooks (lens hook, fixation hook, squint hook, and Tyrrell's hook) are used in operations on the eye; Malgaigne's hooks were used for approximating the pieces of a fractured patella; there are tonsil hooks, uvula hooks, cleft palate hooks, and tracheotomy hooks, used in operations on the tonsils, uvula, soft palate, and the trachea, and polypus hooks for the fixation of aural polypi, etc.; the tenaculum is a sort of cervix uteri hook (*see* Gynæcology, Diagnosis in); the blunt hook of Obstetrics is used to aid the expulsion of the infant in breech cases, etc. (*see* Labour, Management, *Podalic Presentations*), the sharp hook or crotchet to extract a perforated head (*see* Labour, Operations, *Embryotomy*), and Ramsbotham's sharp hook or C. Braun's decapitating hook or key to decapitate the fœtus in impacted shoulder cases (*see* Labour, Operations, *Embryotomy in Trunk Presentations*).

Hookworm Disease.—Ankylostomiasis, miner's anæmia, or uncinariasis. *See* Anchylostomiasis; Parasites (*Strongylidæ*).

Hopkins' Method. *See* Uric Acid (*Detection by ammonium chloride*).

Hoplocephalus. *See* Snake-Bites (*Colubrine Snakes, Elapidæ*).

Hops.—Lupulus. The strobiles of ordinary hops, *Humulus lupulus.* The most important

constituent is *Lupulin*, a liquid alkaloid; but there are also present lupulinic acid, an aromatic volatile oil, resin, and tannin. *Preparations*—1. Tinctura Lupuli. *Dose*—½-1ʒ. 2. Infusum Lupuli. *Dose*—1-2℥. 3. Lupulinum, a bright yellow powder—lupulinic glands—separated from the strobiles of the hops. *Dose*—2-5 grs.

Hops have been used as antispasmodics and nervous sedatives in hysteria, priapism, vesical and renal irritation, and even in delirium tremens. Beer owes its soporific effect partly to the hops contained. The influence of "hop pillows" in nervous insomnia is probably largely imaginary.

Hordeolum. *See* Eyelids, Affections of (*Hordeolum or Stye*).

Horn-pox.—An abortive form of small-pox, in which the vesicles dry up instead of filling about the fifth or sixth day; wart-pox.

Horns. *See* Cornu; Tumours of the Skin (*Benign*).

Horripilation.—The standing up of the hairs on the body from fear or pain (Latin, *horrere*, to bristle, and *pilus*, hair); cutis anserina.

Horrocks' Bags.—Indiarubber bags for the dilatation of the cervix uteri or vagina. *See* Labour, Operations (*Induction of Premature Labour*).

Horrors.—*See* Delirium Tremens (*Visual Hallucinations*).

Horse. *See* Glanders or Farcy; Rheumatism, Chronic (*Comparative Pathology*).

Horse-pox.—A contagious disease of the horse; inoculation of man with the virus gives immunity against small-pox.

Horse-radish. *See* Armoraciæ Radix.

Horsfall Destructor. — A forced-draught furnace for the destruction of refuse, in which a very high temperature is used.

Horsley's Putty.—A hæmostatic substance, consisting of carbolic acid (1 part), oil (2 parts), and wax (7 parts). *See* Brain, Surgery of (*Trephining, Operative Details*).

Hospital Fever. *See* Typhus.

Hospital Gangrene.—Wound phagedæna. *See* Gangrene (*Infective*).

Hospitalism.—The prejudicial effects produced upon the inmates of a hospital by overcrowding (*J. Y. Simpson*).

Hospital Sore Throat. *See* Pharynx, Acute Pharyngitis; Tonsils, Diseases of (*Acute Tonsillitis*).

Hospitals.

Hospitals have been provided for the reception and treatment of the sick from earliest times. Hospitals supported by Government existed in India, Persia, and Arabia before the Christian era. It is declared that the sick were treated in the temple of Æsculapius as far back as 1124 b.c.

Within recent years a very great development has taken place in Scotland by the provision of hospitals for the treatment of infectious diseases, and also for ordinary medical and surgical diseases in cottage hospitals.

There are two distinct classes of hospitals: (1) Those for the treatment of infectious diseases, usually called isolation hospitals, and (2) those for the treatment of non-infectious diseases, such as ordinary medical and surgical diseases. In the first category the hospitals are usually provided under statutory powers by sanitary authorities, and the cost of equipment and upkeep fall upon the public rates.

There is no power by which sanitary authorities can provide hospitals for non-infectious diseases, as Section 66 of the Public Health Act states that any Local Authority may provide, furnish, and maintain hospitals for the use of the inhabitants of their district suffering from *infectious* disease. Space entirely forbids a lengthy description of the general principles to be observed in hospital construction, and it further demands that the description shall only refer to the smaller-sized hospitals suitable for villages and small towns and rural districts.

There will be no reference to maternity hospitals, hospitals connected with medical schools, asylums, workhouses, or with boarding schools; each of these would have required specialised reference to the special exigencies of the type.

There are certain general conditions to be considered in the construction of all houses, whether hospitals, schools, or ordinary dwelling-houses.

The first of these is the site.

Site.—Site has sometimes to be determined under conditions in which the hygienic element is not the most prominent. It should be dry and not malarious, and with an aspect which gives light and cheerfulness. The soil should be open and pervious, such as sand, gravel, and chalk. An underlying bed of clay is most undesirable. A clay soil is distinctly objectionable. Sites on granite, trap, limestone, and sandstone are usually healthy. Where the soil is damp, draining must be resorted to, and in all cases a damp-proof course of concrete or asphalt should cover the surface of the ground enclosed by the walls.

A site with a gentle slope at a moderate elevation facing the south, and protected from prevailing winds is most desirable. A belt of trees,

judiciously planted, may assist in moderating the intensity of winds.

The site should be removed from works which cause smoke and effluvial nuisances. Accessibility by main roads to the district the hospital is to provide for, to the medical attendant, to shops, telegraph and telephone communication, posting establishment, must all be considered. The area needed varies; in infectious diseases hospitals, about an acre for twenty-five to thirty persons is necessary, for non-infectious hospitals much less is required, but the amount will entirely depend on disposition and arrangement of wards, etc.

Construction.—This should be of such a nature as to ensure dryness of foundations, walls, and roof. The materials ordinarily used for walls are stone and brick, but concrete, corrugated iron, wood, are also used. Whatever material is used, the foundation should have a damp-proof course at a level of at least 3 inches above the ground.

Brick walls should be hollow, built with an air space of about 3 inches. The inner surface of the walls to be covered with some impervious material such as Kean's cement, "Adamant" plaster; or glazed bricks may be used. Angles at the junction of ceiling with walls, of walls with floor, of end with side walls, should be rounded off. Cornices, moulding, etc., should be avoided, in brief, all receptacles for dust should be avoided.

Floors should be made preferably of hard wood, oak or teak, or red pine, tongued and grooved, to be afterwards made impermeable by one or other of the carious compositions.

The floor space per bed for infectious diseases hospitals is usually 144 feet with a wall space of 12 feet. The width of ward 25 to 26 feet, giving a total air space of 2000 cubic feet per bed for infectious diseases hospitals. For small-pox cases, 3000 cubic feet are required.

Ventilation under the floors should be provided by openings in the walls at opposite sides. For the roof, slates laid on boarding and felt provide the necessary means for securing dryness and warmth. Rhones and down-spouts are necessary auxiliaries, and the roof-water may be, with advantage, collected in tanks for washing purposes, at the same time simplifying the sewage purification problem.

Water Supply.—This should be, wherever possible, by gravitation, and the water must be abundant and of quality above suspicion. Where sufficient "head" cannot be got, air-motors, rams, water-wheels, turbines, etc., may be necessary to pump up the water from some sufficient source. At least 50 gallons per head are required. Where the quality is not above suspicion, a Pasteur-Chamberland filter should be in operation.

Drainage, Sewage Disposal, and Purification.—In small country hospitals, earth closets might be used, thus simplifying both the question of sewage removal and treatment. For isolation hospitals, the Local Government Board do not favour this system. They should only be used where pressing difficulties of sewage removal occur.

Whether an earth-closet or water-closet is used, it must be cut off from the ward by a passage with cross ventilation.

A wash-down closet with a three-gallon flush is to be preferred. Water-closet soil-pipes, sink and bath wastes, must be constructed on the most approved principles, but space does not permit of desirable details. Sewage, including water from baths and wash-house, must be purified before it is discharged into any stream.

The septic tank system; Ducat's or Dibdin's are the most likely methods now in use.

The methods of sewage treatment referred to depend on bacterial action. In the septic tank the sewage first undergoes anaerobic resolution, water, CO_2, CH_4, and H_3N being formed. The effluent is then run through coke beds, and nitrates are formed, where aerobic action occurs.

In Ducat's system the beds, made of coke breeze, are freely aerated by drain pipes passing into their interior. The sewage is distributed over the top by a sprinkler.

The beds in the latest development are covered over and warmed with hot-water pipes. The purification of sewage resulting is remarkable.

Where earth, peat, or saw-dust closets are used, some form of incinerator will be necessary. It will also be useful for burning poultices, old dressings, sweepings from wards, etc.

Ventilation, Warming, and Lighting.—For small hospitals ventilation is best secured by open fire-places and windows. There should be a window between every bed, and extending from 2 feet 6 inches from the floor to within one foot of the ceiling. One superficial foot of window for every 60 cubic feet of ward space is a fair allowance.

The windows should be double sashed and double glazed. Sometimes a "Hopper" light, hung on hinges, is provided in addition. Openings at the floor level with radiator ventilators, so that the incoming air is heated, are usually provided. Outlets may be further provided by Boyle, Buchan's, or other ridge ventilators. One inch of outlet for every 60 cubic feet of room is a usual ratio allowed.

Warming is provided best by open fire-places, supplemented as necessary by hot-water pipes either high or low (preferable) pressure, or by fire-clay stoves.

Such are the general conditions to be attended to in all hospitals, whether for infectious or non-infectious diseases, to secure abundant light, pure air at a proper temperature, dryness of the building, and prompt removal of offensive matters.

The essential parts of an isolation hospital are :—An administration block, containing nurse and matron's rooms, kitchen, scullery, pantries, larder, linen-store, storeroom, coal-store, nurses' and servants' bedrooms, bathrooms.

The extent of these will entirely depend on the size of the hospital, but cannot be very much curtailed for ordinary sizes.

The wards; and the best arrangement is to have single-storey pavilions for each type of disease.

The sexes to be separated by an intervening nurse's duty-room, with a cooking range and hot-water arrangement.

By means of a closed window the nurse can supervise both wards. A bath on wheels can be kept in the passage or hall, and be used for both wards.

Steam disinfector, laundry and wash-house, ambulance shed, mortuary, coal and stick house, can form another block.

Caretaker's house and discharge rooms. The most suitable arrangement for all these is pavilions running north and south, administration block in front and in middle. Ambulance shed, laundry, etc., in rear. All separated by a 40-feet space.

Non-infectious diseases hospitals should have same conditions as to site, etc. already referred to. Accommodation will depend upon individual circumstances. It may be only a two or three-roomed cottage. One of the most recently established cottage hospitals is arranged thus:

The administration block is central, and the wards form wings in communication by passages with shutting-off doors.

On the ground floor of the administration block is a man's day-room and a woman's day-room, doctor and matron's room, and in the back, kitchen, scullery, and store, etc.

The two main wards are on a north-east line, and have six beds in each separate sexes. There are six private wards, four with one bed in each, and two with three. Above the central building are nurse's and matron's bedrooms, day-rooms, etc.

The floor space for the wards is 90 feet, and the cubic space 1350, and the general internal arrangements are as already described for isolation hospitals.

The operation room is on the ground level, and to the back of the building. The walls and floor are covered with glazed tiles. The operating table is of metal. Wash-hand basins and sink taps can all be wrought by the foot.

Glass tops resting on rubber are provided for the basins. Shelving is of glass on iron supports. A hose is provided for washing and flushing the floors and walls.

The lighting is from the roof. Disinfecting and sterilising rooms are provided for bedding, clothing, and for instruments. This hospital embodies the latest developments in hospital construction.

Rules for Visitors in Infectious Hospitals.—1. No person, unless an officer of the hospital or a member of the hospital committee, shall enter the hospital without a written order, signed by the Clerk or Medical Superintendent, except the Sanitary Inspector of any Local Authorities, who shall have right of entry only when bringing patients to the hospital.

2. Friends of patients will be admitted only when provided with a written order by the Medical Superintendent, except in cases where a fatal result is feared, when the Matron's sanction will be sufficient. Admission, under all circumstances, will be subject to the following conditions :—

(*a*) That not more than two visitors will be admitted at the same time.

(*b*) That each visit shall not exceed five minutes.

(*c*) That no fruit, food, or drink or sweetmeats be given to any patient.

(*d*) That visitors shall not touch patients or their clothes, nor sit on their beds, nor go too near them.

(*e*) That they shall not smoke nor conduct themselves in any way disagreeable to the Matron or patients.

3. Visitors to small-pox cases must have been recently revaccinated, or have had the disease previously, and must cover themselves with an overall dress to be provided for the purpose.

4. All visitors must obey the Matron's injunction in regard to length of visit, modes of preventing infection, etc.

Hot Air. *See* Hydropathy (*Hot-air Applications*).

Hot Bath. *See* Balneology (*Hummum*); Lungs, Vascular Disorders (*Œdema, Treatment*).

Hot Pack. *See* Hydropathy (*Hydropathic Methods, Packs*).

Hot Spots. *See* Physiology, Senses (*Temperature Sense*).

Hour-Glass Contraction. *See* Labour, Retention of Placenta (*Spasmodic Contraction of Uterus*); Stomach, Surgical Affections of (*Hour-Glass Stomach*).

House-Flies. *See* Myiasis.

Housemaid's Knee. *See* Bursæ, Injuries and Diseases of (*Simple Chronic Bursitis*); Knee-Joint, Diseases of (*Chronic Bursitis*).

House Refuse. *See* Sewage and Drainage.

Howard's Method. *See* Asphyxia (*Artificial Respiration, Methods*).

Hum.—A soft, low, continuous sound, like that of a spinning top; such is the venous hum, heard sometimes over a vein, and the amphoric hum, heard over a pulmonary cavity.

Humanised Lymph.—Vaccine lymph from the human subject.

Humanised Milk. *See* Infant Feeding (*Sterilisation*); Milk (*Physiological, Humanising procedures*).

Humerus. *See* Elbow Joint, Injuries and Diseases; Fractures (*Humerus*); Labour, Accidental Complications (*Injuries to Fœtus during Labour*); Shoulder, Diseases and Injuries of (*Congenital Defects of Humerus, Fractures of Humerus, Operation of Excision of Head of Humerus, etc.*).

Humidity. *See* Meteorology (*Humidity*).

Hummun. *See* Balneology (*Hot-air Baths*).

Humoral Pathology. *See* Medicine, History of (*Hippocratic School*).

Humulus Lupulus. *See* Hops.

Hunger. *See* Hypnotism (*Experimental Phenomena*); Stomach and Duodenum, Diseases of (*General Symptomatology*).

Hunterian Chancre.—The hard sore of syphilis, described by John Hunter. *See* Syphilis (*Acquired, Primary,* Pathology of).

Hunter's Canal. *See* Arteries, Ligature of (*Ligature of the Femoral Artery*).

Hunter's Method. *See* Aneurysm (*Treatment, Ligature at some distance above the sac*).

Huntingdon's Chorea. *See* Chorea (*Hereditary Adult Chorea*).

Hunyadi Janos Water. *See* Balneology (*Austria, Sulphated Waters*).

Hutchinson's Teeth.—The notched condition of the *permanent* teeth seen in hereditary syphilis, and first described by Mr. Jonathan Hutchinson. *See* Syphilis (*Syphilis in Children, Hereditary*).

Hutchinson's Triad.—The term applied by A. Fournier to three of the chief signs of hereditary syphilis, viz. interstitial keratitis, malformations of the teeth, and deafness; for these were specially described by Mr. Jonathan Hutchinson, and he showed their frequent coincidence and relationship with hereditary syphilis.

Hyaline Cartilage. *See* Physiology, Tissues (*Cartilage, Varieties*).

Hyaline Casts. *See* Nephritis (Chronic, *Urine, Characters of*); Urine, Pathological Changes in (*Sediments, Casts, Hyaline*).

Hyaline Degeneration.—A pathological change, affective connective tissue, and resembling waxy degeneration (Gr. ὕαλος, glass); it is most frequently met with in the small arteries and capillaries of the spleen, kidney, brain, and spinal cord; and it is characterised by swelling of the tissue elements, which also become translucent, homogeneous, and less easily digested. The hyaline substance is not specially stained by iodine or the aniline dyes; it resembles keratin in its resistance to chemical agents. It is met with in acute infectious fevers and septicæmia, as well as in chronic toxic states, such as chronic nephritis. It may be an early stage of waxy degeneration, and it is not infrequently followed by calcification. There is a necrotic change in muscle which in some degree resembles hyaline degeneration (*e.g.* in typhoid fever), but it is not the same process. *See* Heart, Myocardium and Endocardium (*Pathology, Morbid Processes, Degenerations*); Insanity, Pathology of (*Pathological Anatomy, Cerebral Blood-Vessels*); Lardaceous Degeneration; Muscles, Diseases of (*Degenerations, Hyaline*).

Hyalitis.—Inflammation of the vitreous humour. *See* Choroid, Diseases of (*Suppurative Choroiditis, Pathology*).

Hyaloid.—Belonging to the vitreous humour, *e.g.* the hyaloid membrane or thin fibrous capsule surrounding the vitreous, the hyaloid or central artery of the vitreous which occasionally persists after birth as a malformation. *See* Embryology, Human (*Embryo in the Sixth Week*); Physiology, The Senses (*Vision, Dioptric Mechanism*).

Hyalophagia.—The insane desire to chew and swallow glass.

Hyaloplasm.—The more homogeneous part of the protoplasm of a cell. *See* Physiology, The Cell (*Movements*).

Hybrid.—Having a mixed character, *e.g.* hybrid measles or scarlet fever. *See* Measles (*Aberrant Forms*).

Hybridism.—The condition of being hybrid, or the production of hybrids; cross-breeding. *See* Heredity (*Cross-Breeding*).

Hydatid Disease.

See also Bladder, Injuries and Diseases (*Foreign Bodies in Ureter*); Brain, Cysts (*Hydatid*); Diaphragm, Surgical Affections of (*Subphrenic Abscess, Causes*); Gall-Bladder and Bile Ducts, Diseases of (*Distension of Gall-Bladder, Diagnosis*); Hæmoptysis (*Etiology, Hydatids of Lung*); Heart, Myocardium and Endocardium (*General Pathology, Etiology, Parasitic Agents*); Kidney, Surgical Affections of (*Movable Kidney, Diagnosis*); Kidney, Surgical Affections of (*Hydatid Cysts*); Labour, Prolonged (*Pelvic Deformities, Hydatids of Pelvic bones*); Liver (*Perihepatitis, Secondary*); Liver (*Tropical Abscess, Diagnosis*); Liver (*Hydatids of*); Lungs, Vascular Disorders (*Embolism from Hydatid Cysts*); Lungs, Abscess of; Mammary Gland, Diseases of (*Cysts*); Mediastinum (*Growths, Hydatid Cysts*); Mouth, Diseases of (*Floor of Mouth, Hydatids*); Neck, Region of (*Hydatid Cysts*); Orbit, Diseases of (*Parasitic Cysts*); Pericardium, Diseases of (*New Growths*); Peritoneum, Tumours of; Pleura, Affections of, Surgical; Spleen, Surgery of (*Echinococcus Cysts*); Tongue (*Cysts of*).

Syn.: *Echinococcus Disease*

Corresponding to each of the adult cestodes there is an immature form or larva, occurring usually in the tissues of an intermediate host, and developed from an ovum of the adult worm. The larva at a certain phase of its life-history exhibits the development of a "caudal vesicle," bladder, or cyst, and from this vesicle one or more heads, or scolices, may arise, which again develop, on gaining a suitable host, into the adult worm. In *Cysticercus cellulosæ*, the larva of *Tænia solium*, one head is formed from each bladder-wall. In *Cœnurus, e.g. C. cerebralis* (the cystic stage of *T. cœnurus*), which causes "gid" or "staggers" in sheep, many head are budded out from the wall of the bladder, which is unilocular. The larva of *T. echinococcus* is also polycephalous, but the heads originate from the walls of special vesicles inside the primary bladder. The larvæ of *T. echinococcus* are termed echinococci, or hydatids, and their presence in the body constitutes the condition known as hydatid disease.

Tænia Echinococcus is a cestode belonging to the family of *Tæniadæ*. It is 4-5 mm. in length, and consists of only three or four segments or proglottides in addition to the head or scolex, which has a transverse diameter of 0·3 mm. The head possesses four muscular suckers and a rostellum with two circlets of hooks, each circlet consisting of 14-25 hooks, which measure from 0·03 to 0·04 mm., those of the inner series being the larger (Leuckart). The hooks have stout root processes. The first segment is small, the second shows sexual organs, but the terminal segment, which is 0·6 mm. broad by 2 mm. long, and thus constitutes $\frac{1}{2}$-$\frac{1}{3}$ of the whole worm, is the only segment which is sexually mature. Like the proglottides of most other teniæ the terminal segment has a single marginal genital pore. The cirrhus (penis) and the cirrhus pouch are well developed, and the testicles number about sixty. The female organs consist of vagina, receptaculum seminis, oviduct leading to two ovaries, yolk gland, and uterus with short lateral ramifications. The uterus contains about five hundred ova, which are of about the same size as the ova of other cestodes (0·02-0·03 mm.), though the hard shell is somewhat thinner.

The *habitat* of *T. echinococcus* is the small intestine, where the white mature proglottides of hundreds or thousands of the worms may be seen protruding between the villi and showing lively movement. The dog is the chief host of this tænia, which is easily distinguished by its minute size from the other intestinal cestodes of the dog, and the tænia has also been found in the wolf (Cobbold), jackal, and a puma (*Felis concolor*). No other animal appears to naturally harbour the adult worm, and experiments have shown that *T. echinococcus* does not develop in the intestine of cats (Peiper), foxes, or rabbits (Leuckart), nor has it ever been found in the intestine of the human subject. The adult worm is developed from the scolices of the cystic larva, and dogs are naturally infected by eating the infected organs of an intermediate host. The metamorphosis of larval scolices into mature tæniæ occurs in 4-11 weeks, but as a rule is not complete till the seventh week (Leuckart).

Hydatids or Echinococci.—*Hosts.*—Mammals are almost without exception the only hosts. Sheep, oxen, swine, and man are the chief hosts, whilst the horse, kangaroo, goat, and other mammals are less often affected. Although the cystic worm in the human subject is termed *E. hominis*, and in the lower animals is called *E. veterinorum*, yet the specific identity of these two forms has been proved.

Mode of Infection.—The mature proglottides or ova of *T. echinococcus* are deposited with the dog's fæces on the pasture, and the hard shell of the ovum to some extent protects the embryo inside. Herbivora are infected by feeding on the contaminated pasture. For the human subject, too, the dog is the sole source of infection. The licking of the human face and hands by dogs is often the means of infection, for the tænia may get on to the dog's tongue by migration, or in consequence of the dog's vomiting. There may be ova or proglottides of the tænia on the dog's muzzle too, because of the dog snuffing at its own anus, or the ani or fæces of other dogs. Ova may also be deposited on the floor from the

dog's muzzle or hindquarters, and toys, bits of bread, etc., may thus be contaminated. Again, the dog's fæces may be deposited in the neighbourhood of springs or pumps, and any proglottides or ova will tend to be preserved by the constant moisture, and will eventually reach the stomach in the drinking-water (Mosler). Many Australian writers assert that in their country infection is probably mainly due to drinking contaminated water. The eating either of uncooked vegetables, such as salad, or of berries and other fruits which grow near the ground, may also be a source of danger.

Development.—If the ova of *T. echinococcus* enter the stomach their shells are digested, and the asexual six-hooked embryos are carried to the intestines, and bore their way (Leuckart) through the intestinal walls. The embryos thus reach venous or lymphatic channels, and are distributed throughout the body. As the embryos are mainly distributed by the portal system the liver is the most frequently affected organ. They may, however, be carried by the lymphatics to the mesenteric glands or peritoneum, and may gain the pulmonary or systemic vessels not only by traversing the liver, but also by the thoracic duct.

Having reached its destination in any part of the body the embryo loses its hooks, enlarges, becomes converted into a cyst, and excites a chronic inflammation of the surrounding tissues. The developmental changes were most thoroughly investigated by Naunyn and Leuckart. In the liver of a pig, four weeks after the ingestion of ova, Leuckart found small nodules each about 1 mm. in diameter. In the interior of each of these was the altered embryo, which is globular and resembles a mammalian egg, there being a thick, homogeneous, transparent outer zone enclosing coarsely granular material. Eight weeks after ingestion the larva had doubled in size, the granular material was clearer owing to partial liquefaction, and the fluid having collected towards the centre a cyst was formed, whilst the granular material situated more peripherally, just inside the homogeneous outer zone, had become differentiated into a definite layer, termed the parenchymatous or germinal layer, or endocyst. At this stage the endocyst consisted chiefly of granules and pale, drop-like cells, which sometimes showed stellate ramifications. When nineteen weeks old the cyst measured about 1 cm. in diameter, the homogeneous outer zone had attained a thickness of 0·2 mm., was distinctly laminated, and is called the external laminated layer, ectocyst, or cuticle. The ectocyst is formed by deposition in successive layers from the parenchymatous layer. The latter layer at this stage was not more than 0·12 mm. thick, yet could be differentiated into two layers—an inner consisting of small, clear droplets, and an outer layer of a more cellular nature—whilst there were highly refractile granules and calcareous corpuscles between the cells. External to the ectocyst is an adventitious fibrous tissue capsule formed by proliferation of the surrounding connective tissues.

Development of Brood-capsules.—When the cyst is about the size of a hazel-nut small elevations arise on the parenchymatous layer by cellular proliferation. The centre of each of these elevations becomes hollowed out, and the vesicle, or "brood-capsule," so formed is bounded by two layers similar to those of the parent cyst; but in the brood-capsule the laminated layer lies internal to the parenchymatous layer. The older hydatids may contain thousands of brood-capsules, none of which, however, exceed 1·5-2 mm. in diameter.

Formation of Scolices.—Each scolex originates as a diverticulum from the wall of a brood-capsule, the cavity of the latter being continuous with the canal-like cavity of the diverticulum, which is, therefore, lined by a cuticle. At the distal end of this diverticulum, or rudimentary head, hooks and four suckers appear. The scolex is markedly contractile, and eventually becomes invaginated so as to project into the cavity of the brood-capsule; and the cuticle which formerly lined the canal is now the outer covering of the scolex, whilst the suckers and hooks are external too. The scolex, which is never more than 0·3 mm. in length, is now a solid cylindrical mass, attached posteriorly by a "stalk" to the wall of the brood-capsule; but the anterior or cephalic half of the scolex often becomes invaginated into the posterior part, so that the scolex has then a spherical shape. The hooks are smaller than those of the adult tænia, from which they are also distinguished by their small, slender root processes. Leuckart maintains that the bladder wall, brood-capsules, and scolices are all in direct structural continuity with one another, and that it is only as a result of exposure to deleterious influences, *e.g.* contact with water or death of the host, that the brood-capsules burst, when the scolices are either at once set free, or remain for a time attached to the shrivelled wall of the brood-capsule.

In many instances, and especially in the case of *E. veterinorum*, the larva undergoes no further developmental changes (acephalocyst), and growing but slowly it rarely exceeds an orange in size, and is either globular or irregular in outline, according to the resistance of the surrounding tissues. The ectocyst increases in thickness; and the adventitious capsule is often 5 mm. thick and very firm.

Formation of daughter-cysts, which have the same structure and function as the mother-cyst.—1. *E. exogenus* or *E. granulosus*, where daughter-cysts lie outside the mother-bladder. These daughter-cysts originate as buds in

the deeper layers of the maternal ectocyst (Leuckart), the buds enlarge, push forward, and finally burst through the superficial layers of the maternal ectocyst. They form an external cuticle for themselves, their central portion becomes liquefied just as in the case of the parent cyst, and the daughter-cyst may finally be completely separated from the parent cyst by the fibrous capsule of the former. *E. exogenus* is most common in the domestic mammals, especially the pig; whilst in man it is rarer, and has been found mainly in the bones and on the peritoneum, though in the liver or other organs also.

2. *E. endogenus* or *E. hydatidosus*, in which daughter-cysts lie within the parent cyst. The (0·1-4 mm. in diameter), enclosing clear granular substance, and separated from one another by a fibrous stroma, so that the appearance resembles that of a colloid carcinoma. The multilocular echinococcus has a tendency to degenerate at its centre and to become calcified. The line of separation between the echinococcus and the surrounding liver tissue is very irregular, and the liver is often bile-stained. This variety has also been found affecting the lungs, peritoneum, and brain, but is very rare in other situations. The spaces between the fibrous stroma, like the bladders of other echinococci, possess a laminated cuticle, but scolices are seldom developed. *E. multilocularis* probably originates, not in consequence of infection by many embryos, but

Fig. 1.—Adult Tænia echinococcus (×12). Leuckart.

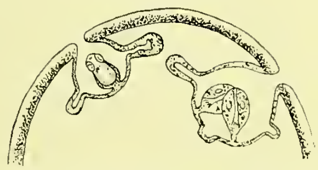


Fig. 2.—Diagrammatic representation of a proliferating echinococcus. Leuckart.

Fig. 3.—Hook of Echinococcus veterinorum (×600). Leuckart.

Fig. 4. — Echinococcus-head, with the anterior part of the head invaginated (× 90). Leuckart.

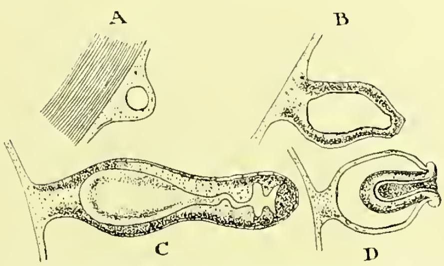


Fig. 5.—Development of brood-capsules (A) and of the appended heads; (B) first rudiment of the head; (C) further development; (D) invagination (× 90). Leuckhart.

daughter-cysts may be few in number and of globular form, or may number some hundreds or thousands, and then from mutual compression exhibit irregular forms. They originate in retrograde metamorphosis of scolices and brood-capsules, which thereby assume the structure and functions of daughter-bladders. If they be formed from brood-capsules the superficial parenchyma of the latter disappears, the scolices lose their form, and ultimately become a layer which is evenly distributed over the *internal* surface of the cuticle, thus forming a new parenchymatous layer. Daughter-cysts may also be formed from folds of the maternal parenchyma, portions of which form a cuticle, and become hollowed out internally in the same way as exogenous cysts.

In either of these varieties granddaughter-cysts may arise from the daughter-cysts just as the latter do from the mother-cyst.

3. *E. multilocularis* is usually found in the human liver, and varies in size from that of a pea to that of a child's head. On section it is found to consist of numerous small spaces from one embryo by repeated exogenous proliferation, a mode of development somewhat similar to that of the racemose forms of echinococci seen in the livers of cattle.

The laminated ectocyst is highly elastic, and when incised curls upon itself. It possesses considerable capacity of imbibition, and contains chitin and a small amount of hyalin (Hoppe-Seyler).

Hydatid fluid is colourless, transparent, of neutral alkaline or faintly acid reaction, and has a specific gravity varying usually from 1009 to 1015. It usually contains 97-98 per cent H_2O, 0·5-0·8 per cent NaCl, succinic acid and inosite. The fluid may contain other constituents owing to the absorptive power of the ectocyst; the fluid of a hydatid in the liver may contain grape sugar, leucin, tyrosin, cholesterin, cystin, and hæmatoidin, whereas in the kidney it may contain crystals of uric acid, oxalic acid, triple phosphates, and other earthy salts. The normal fluid never contains any coagulable albumin, which can as a rule, however, be detected if the cyst wall is inflamed or has been previously

punctured. In the latter case the presence of albumin is possibly due to functional changes induced in the hydatid by the puncture.

Changes liable to occur in the Hydatid Cyst.—The echinococcus often dies spontaneously. Degenerate changes commence just outside the ectocyst; the fluid becomes opaque, coloured, albuminous, and reduced in amount; the scolices fall off, and the hyatid is represented by a pulpy débris which still contains the hooks and possibly fragments of ectocyst, and which, together with the capsule, may undergo calcareous infiltration. Partial ossification has been observed in rare cases. Death of the larva may also be caused by evacuation of the fluid it contains, or by suppuration commencing round the cyst and spreading to its interior. Rupture of the cyst is another possible occurrence, either as a result of trauma or of atrophy of surrounding structures from pressure.

Geographical Distribution.—Statistics from Iceland represent 1·2-3 per cent of the population as affected with hydatids. Hydatids do not occur in the Faröe Islands (Davidson). In German-speaking countries patients with this disease formed 0·03 per cent of a total 500,000 cases treated in hospital (Neisser), but the disease is comparatively rare in Southern and Central Germany, while more common in the north, especially in Mecklenburg and Pomerania. For example, in Rostock there is one case of hydatid disease for every 1056 inhabitants. The following figures illustrate the frequency of the disease in different parts of Germany (Peiper):—

		Hydatids in total Autopsies.
Kiel	(1872-1887)	0·19 per cent.
Munich	(1854-1887)	0·25 ,,
Jena	(1866-1887)	0·84 ,,
Greifswald	(1862-1894)	1·50 ,,
Rostock	(1861-1883)	2·43 ,,

In Switzerland ordinary hydatids are rare, occurring in 0·13 per cent of all autopsies. In Vienna (1892-1897) hydatids occurred in 0·02 per cent of hospital patients. In Dalmatia, however, the disease appears to be more common than in any other part of Europe. In Servia 0·02 per cent of hospital patients are affected. In Belgium and Denmark the disease is rare, only four cases are reported from Norway, in Italy and France hydatids are more often seen. In Great Britain hydatid disease is uncommon, but it is more frequent in England than in Scotland (Murchison). In *St. Thomas's Hospital Reports* 1894-1898 there are 19 cases; in *Middlesex Hospital Reports* 1893-1897 there are 15 cases; in *Westminster Hospital Reports* 1889-1898 only 10 cases; and in *Edinburgh Hospital Reports* 1891-1897, 11 cases are recorded. In South Africa, Egypt, China, and India, hydatid disease is rare. Osler could find records of only eighty-five cases in the United States and Canada up to July 1891. In the Argentine Republic the disease is common, but very rare in other parts of South America. The disease is common in some parts of Australia, especially in Victoria and South Australia. The average death-rate per 100,000 from this cause during the period 1882-1886 was as follows:—Victoria, 5·79; South Australia, 3·54; New South Wales, 2·05; Queensland, 1·29. In Tasmania hydatids are rather common, giving a death-rate of 2·83 per 100,000, but in New Zealand the corresponding mortality is only 0·66 per 100,000 (1882-88, Davidson).

Thus the disease is most frequent in Iceland, Australia, Dalmatia, and parts of Northern Germany.

Posselt has recently collected records of 215 cases of multilocular echinococcus. Their geographical distribution, which is very remarkable, is as follows:—1. Bavaria 56, Austria (the majority in the Tyrol) 30, Switzerland 27, Würtemberg 25, Baden 3, etc., in short 147 cases within a circumscribed area; 8 cases in districts adjacent to this area; 40 cases in the two Russian territories of Kasan and Moscow. 2. 20 cases which occurred irregularly in various countries. No case has occurred in Great Britain. There is as yet no definite proof that multilocular echinococcus is derived from a special variety of *T. echinococcus.*

The following factors favour the occurrence of hydatid disease:—

I. A high percentage of dogs infected with *T. echinococcus.* 28 per cent of dogs were said to be affected in Iceland, and 6 per cent in Copenhagen (Krabbe). The tænia is a common parasite in Mecklenburg (Madelung), and was found in 40 per cent of unregistered dogs in Australia (Thomas). In England "probably at least 1 per cent of our dogs harbour the mature tape-worm" (Cobbold), whilst in North America it is a rare parasite (Osler).

The actual frequency of the dog is of less importance. For example, the ratio of dogs to individuals is 1 : 11 in Iceland, 1 : 18 in Mecklenburg (Madelung), and the ratio of registered dogs to persons in Australia is 1 : 23, whilst the registered dogs constitute only a fractional portion of the total (Graham), yet the ratio is 1 : 27·8 in Pomerania, 1 : 22 in France, and 1 : 15 in Belgium.

II. Circumstances facilitating the transmission of the ova to the human subject:—

The disease occurs most frequently in those who are brought much in contact with dogs, *e.g.* Australian shepherds, and is more common in country parts than in towns. The disease is common too in Iceland, where during the long winters the dogs live in the same room with the people, many of whom are so careless that instead of washing their dishes they permit them to be licked clean by the dogs (Leuckart).

III. Many domestic mammals, and, still

more important, the frequent occurrence of *E. veterinorum* in them.—For every hundred persons in Australia there were in 1892, 300 cattle, 3000 sheep (Verco and Stirling), in Iceland there were in 1883, 46 cattle, 488 sheep (Krabbe), in Mecklenburg-Schwerin there were in 1884, 47·3 cattle, 164·4 sheep, 39·5 swine (Madelung), and in Pomerania there were 168·4 sheep to every 100 persons; whereas for every 100 persons in Italy there were 16·8 cattle, 30·2 sheep, 4·1 swine, in France there were 30·4 cattle, 59·8 sheep, 14·8 swine, in United Kingdom there were 28·2 cattle, 86·4 sheep, 8·2 swine (Madelung), and in these countries *E. hominis* is comparatively rare.

Again, the prevalence of hydatid disease in man is proportionate to the frequency of the disease in the domestic animals. Peiper has shown this to be the case in the various districts of Mecklenburg, *e.g.* in Greifswald, where hydatids are common in man, 64·58 per cent of cattle, 51·02 per cent of sheep, and 4·93 per cent of swine are affected. In other parts of Germany not only man but the domestic animals too are comparatively rarely affected, *e.g.* in Karlsruhe 2·90 per cent of cattle, 2·24 per cent of sheep, and 0·66 per cent of swine have hydatids. In Australia about 44 per cent, and in the Argentine Republic, 30 per cent of sheep are affected, and in Iceland the disease is common both in sheep and cattle.

The occurrence of *E. veterinorum* is favoured by a mild damp climate, in consequence of which the ova of *T. echinococcus* may lie for months on the pastures and still be capable of development. It is the animals kept at pasture during the summer which are most frequently infected.

IV. Circumstances facilitating the metamorphosis of the larval scolices into adult tæniæ.—The more frequent the occurrence of *E. veterinorum* in the animals slaughtered the greater are the risks of dogs becoming infected. For the diseased viscera are often carelessly disposed of, or even employed to feed the dogs. In the absence of proper slaughter-houses, as in some parts of Mecklenburg until comparatively recently, dogs are likely to acquire infection.

Hydatid disease in relation to the *occupation* of the individual.—It is most common in those who are much in contact with dogs, *e.g.* shepherds. The possible modes of infection are, however, very numerous, and the disease has been observed in joiners, tailors, and others, though very rarely in seafaring individuals.

Sex.—The relative frequency in the two sexes depends largely on their occupations and pursuits. In Iceland the disease is more common in women, as Finsen in 255 cases found 181 in the female sex. Neisser found 436 cases in females out of a total of 669; Davaine and Peiper hold that the sexes are equally affected; but in Australia hydatids are more common in men, the ratio being 100 : 77 (Verco and Stirling).

Age.—Hydatids occur at almost any age, but are rare in children. Neisser gives the following figures:—0 to 10th year, 4·8 per cent; 11th to 20th year, 13·2 per cent; 21st to 30th year, 30·8 per cent; 31st to 40th year, 24·6 per cent; 41st to 50th year, 15·2 per cent; 51st to 60th year, 6·2 per cent. Hence the disease is most commonly seen between the ages of twenty-one and forty, and is rare after sixty.

Anatomical Distribution.—In swine the liver is more often affected than the lungs (18 : 2·3), in sheep the liver is less often affected than the lungs (14 : 21), whilst in cattle these organs are about equally affected (7·5 : 8·2) (Peiper).

According to Neisser's statistics of 900 cases, the distribution in the human subject was as follows:—Liver 451, lung 67, spleen 28, pleura 17, heart or vessels 29, cranial cavity 68, spinal canal 13, kidney 80, pelvis 36, female genitals or mamma 44, bones 28, face orbit or mouth 21, other parts 18. As a rule only one organ is affected, and in fifty per cent of cases it is the liver.

In some cases, however, there are *multiple echinococci*. In animals these are of common occurrence, and of comparatively small size. In man the liver usually contains one echinococcus, whilst there are echinococci in other situations too. The peritoneum is one of the sites specially affected, for here a solitary echinococcus is rare, but several hundreds are not uncommon. The cause of multiple echinococci is variable. They are, however, usually referred to infection by numerous embryos at one time, to repeated infection, or lastly, and especially in the case of the peritoneum, to development from daughter-bladders, brood-capsules, or scolices which have escaped from the parent-cyst in consequence of tapping or rupture.

An illustrative case has been recently recorded by Subbotic. At the post-mortem on this patient, a woman aged 65, there were in the right lung five echinococci each the size of a pea; the liver was enlarged to four or five times its normal size. It contained an echinococcus as large as a child's head, with daughter-bladders the size of apples, two echinococci each as large as a fist, and one other the size of an apple. The omentum was adherent to the liver, and contained numerous echinococci varying in size from a hazel-nut to a walnut, and of these the smaller ones were degenerated. There were also echinococci in the appendices epiploicæ of the descending colon, in the pouch of Douglas, in the fundus of the uterus, and in the appendix vermiformis.

General Symptomatology.—The symptoms of echinococci are dependent on their site and size, and on the inflammatory and pressure effects on neighbouring viscera. In many situations, *e.g.* the liver, if the cyst does not interfere with any

vital function, and does not injuriously affect the neighbouring viscera, it may produce no symptoms, and even when the size of a child's head may only be detected post-mortem. In other instances the hydatid, as it grows slowly, may have existed for two to thirty years, and be of considerable size before the patient seeks advice; yet the actual duration of the disease is always uncertain, for the date of infection can never be accurately determined. The condition is characterised by the slow and, as a rule, steady growth of the cyst forming a globular, painless, elastic fluctuating swelling, which is not accompanied by fever or cachexia. Fluctuation can only be determined when the echinococcus approaches the surface, and in such cases percussion may occasionally elicit the "hydatid thrill" or fremitus. This "thrill," however, can seldom be obtained, does not necessarily indicate the presence of daughter-cysts, and is not at all characteristic, as it may be elicited in ascitic fluid, or in any tense cyst such as a parovarian cyst. The echinococcus may attain an enormous size, and the pressure effects, which are similar to those of any other cystic tumour, are often very considerable. They vary, of course, as to the organs involved; echinococci of the liver produce embarrassment of the stomach, intestines, heart, or lungs; echinococci of the pleura or lung cause symptoms resembling those of pleuritic effusion or pulmonary tumour; echinococci of the pelvis cause symptoms referable to the rectum, bladder, etc. Erosion of bone may occur from pressure. If the disease affect the brain, spinal cord, or orbit, symptoms will arise even whilst the echinococcus is small.

The symptoms are also dependent on the degree of inflammatory reaction excited. If this be slight it results merely in the formation of a fibrous capsule, but it may, however, cause a chronic inflammation in the affected organ, *e.g.* hepatic cirrhosis, with consequent functional disturbance. Or the inflammation may proceed to suppuration with the formation of a localised abscess. This may rupture and so cause purulent inflammation of a serous cavity, or death may result from pyæmia.

In other cases, and usually in consequence of traumatism, a non-suppurating cyst may rupture. The gravity of this occurrence depends on the site into which the contents of the cyst escape. Rupture of a large cyst into the pleura, trachea, bronchi or peritoneum, is usually fatal; rupture into the alimentary or urinary tract, into the vagina or bile ducts, is much less serious, while rupture through the skin is the most favouable of all forms of rupture.

Echinococcus of the liver forms from 50 per cent (Neisser) to 66·9 per cent (Mosler and Peiper) of all hydatids. It is usually single, situated in the right lobe, and of slow growth. If small it may cause no symptoms, seldom produce jaundice, though more often some cirrhosis, and the patient may be cured by death of the echinococcus as so often happens, or by rupture into the bile ducts. There may be dropsy, however, from pressure on the vena cava by a small hydatid, so situated as not to be detectable on examination. The cyst is, however, often of large size, may occupy the greater part of the abdomen, and is often adherent to other abdominal viscera which become displaced. Such a cyst produces discomfort, a dragging sensation in the abdomen, jaundice and dropsy being unusual. If the hydatid be of only moderate size there will be a visible swelling mainly in the right hypochondrium, smooth on the surface, usually yielding fluctuation, and sometimes a hydatic thrill. Over this swelling there is a dull note continuous with that of the liver. If suppuration occur in such a cyst the symptoms will closely simulate those of abscess of the liver; but if the cyst merely increase in size the abdomen becomes enormously distended, and pressure symptoms become well marked—dyspepsia, constipation, dyspnœa, palpitation, etc. Rupture of such a hydatid may occur into the intestines, with either a subsequent cure, or less frequently with suppuration in the cyst. Rupture into the stomach, intestines, or bile ducts is indicated possibly by some diminution in the size of the swelling, but more certainly by the occurrence in the vomit or fæces of portions of cyst wall, hooks, daughter-cysts ("skins"), etc., whilst rupture into the bile ducts may cause biliary colic. Rupture into the peritoneum is usually followed by fatal peritonitis, rupture into the vena cava causes death from pulmonary embolisms, whilst rupture into the kidney or through the skin is less serious.

If the echinococcus be situated towards the convex surface of the liver its enlargement occurs mainly upwards into the thorax. This variety is often spoken of as a "*subphrenic hydatid*," and in association with it there is often pleuritic effusion on the right side. The growing cyst displaces the diaphragm, right lung and heart, or may grow through the diaphragm, and more or less fill the right pleura.[1] The symptoms and signs of echinococci extending into the thorax from the liver are chiefly those of pulmonary hydatid disease.

Multilocular Echinococcus.—If symptoms be caused, as is not always the case, they differ from those of ordinary echinococci of the liver. In the former condition, which has never yet been seen before puberty, the first symptoms are mainly gastric, and are followed by jaundice which is usually very severe. The liver

[1] Lenhartz has reported a case where by means of the Röntgen rays the diaphragm was found to bulge upwards to the level of the second rib (*Münch. med. Wchnschr.* 1899, p. 1696).

becomes much enlarged, is firm, hard, and often irregular on the surface, whilst it is not much altered in shape, and is not tender. There is often progressive enlargement of the spleen. The state of nutrition and general health of the individual remain remarkably good for a long time. At a later stage there are often hæmorrhages from various sites, and some fever from degeneration at the centre of the diseased area, and towards the end there is often very marked ascites. Multilocular echinococcus has been mistaken for carcinoma or hypertrophic cirrhosis of the liver, but it mainly occurs between the ages of twenty-seven and fifty, develops slowly, and does not cause cachexia.

Pulmonary Echinococci. — 1. In the lungs (7·4, Neisser). The echinococcus is usually single, may be situated in any part, but more often on the right side than the left (25 : 11, Neisser), and is as a rule in the lower lobe. The adventitious capsule, as is always the case when the echinococcus is well protected, is extremely thin, and the cyst not infrequently occupies the greater part of one-half of the thoracic cavity. The pressure effects on the heart and lungs are slowly developed, for the cyst grows slowly and insidiously, and the patient who is apparently in good health merely suffers from dyspnœa on exertion. The physical signs are those of a solid tumour or a pleuritic effusion—impairment of movement, possibly bulging of the chest wall, dulness, with absence of vocal fremitus, breath sounds and resonance. The cyst while still of moderate size often ruptures into the bronchi.

Reid narrates the case of a man, æt. 37, who had never previously been ill, and who whilst in perfect health was suddenly seized with a violent fit of coughing and expectoration of over half a pint muco-pus. There was a patch of dulness below the angle of the left scapula with absence of breath sounds. The sputum contained abundant hooklets. The patient was operated on and recovered.

In some cases there is fever from secondary pleurisy, and if there be hæmoptysis too from rupture of the cyst, the symptoms are not unlike those of phthisis.

In spite of rupture into the bronchi there may be neither hooks nor portions of cyst wall detectable in the sputum.

Reid records the case of a man, æt. 32, who six years previously had typhoid fever and pneumonia. The patient had ever since suffered from cough; he had repeated violent attacks of coughing with expectoration of large quantities of frothy, watery, slightly blood-stained and somewhat offensive sputum. Microscopic examination gave no evidence of hydatid disease; the pulmonary hydatid finally began to suppurate, and the patient recovered after operation.

In cases where a hydatid of the liver has perforated into the lung, the sputum may be of an ochre-yellow colour. Lenhartz in such a sputum found cholesterin, bilirubin and fatty granules.

2. In the pleura (1·9 per cent, Neisser). The echinococcus is either primary or else secondary owing to it invading the pleura from elsewhere, usually from the liver. The pleura itself usually constitutes the adventitious capsule. The signs are those of a pleuritic effusion, but the dulness is often localised, with its upper limit dome-shaped, and is not altered by change of position; whilst suppuration of the cyst produces symptoms similar to those of empyema.

A patient suffering from a pulmonary echinococcus may be cured by death of the parasite, rarely by rupture into the bronchi or alimentary tract, or rupture through the parietes. The disease is often fatal, death resulting from rupture into the bronchi or pulmonary artery, tuberculosis or gangrene of the lungs, or marasmus.

Echinococci of the Urinary System. — The kidney is affected in 8·9 per cent (Neisser)—2·8 per cent (Mosler and Peiper) of all cases of hydatids. The echinococcus is usually unilateral and situated primarily in the cortex, but as the cyst increases in size the whole kidney may become involved, and a large, firm, smooth swelling may be produced in one or other side of the abdomen. Symptoms may be entirely absent, and the condition may last for fifteen to twenty years, and not cause much inconvenience. The cyst may, however, attain an enormous size, when there will be pressure symptoms, and adhesions will as a rule form between the cyst and the bowel, diaphragm, bladder, or spleen. Rupture of the cyst into the pelvis of the kidney may occur at any time, and is a somewhat favourable indication. Manasse has collected 51 cases from the literature where such rupture occurred. A case recorded by Wiesinger furnishes a good illustration. The patient had for two years a hydatid of the left kidney. He fell on his left side, the cyst ruptured, and blood and numerous hooks appeared in the urine. In consequence of rupture the abdominal swelling may diminish somewhat in size, and there is often at the same time renal colic. The urine rarely contains blood, but becomes cloudy or milky, contains pus cells, various crystals, hooks, portions of membrane, small daughter-cysts or brood-capsules, the latter being seen as transparent globules no larger than grape stones. The escape of cyst contents per urethram may continue for only a few days, or may recur from time to time for more than twenty years. By cystoscopy it was possible in Manasse's case to ascertain that the orifice of the right ureter was enlarged to four times its normal size, and that thence the hydatids entered the bladder. If symptoms of pyelitis or suppuration of the cyst

should occur the condition becomes more serious.

Echinococci of the Bladder.—Manasse cites two cases, one of them having symptoms of severe cystitis and passage of hydatids per urethram. The latter symptom is, he says, the only characteristic sign of echinococci of the lower urinary passages in general, although there may be difficulty in micturition and defæcation, and although one may detect a fluctuating swelling. Manasse states that it is often difficult to determine whether the cyst is a primary hydatid of the bladder, or a hydatid which has developed in the subperitoneal connective tissue above the prostate, and which has become adherent to and possibly ruptured into the bladder.

Echinococci of the Spleen.—Three per cent (Neisser), 2·87 per cent (Mosler and Peiper). Trinkler in 1894 collected from the literature seventy cases occurring during the previous fifteen years. The cyst usually attains a large size in the course of time, and there is not infrequently an echinococcus in some other organ. The spleen itself is usually atrophied, less frequently hypertrophied. There are no special symptoms produced, but there is eventually a large fluctuating swelling which is either confined to the left hypochondrium or extends into other regions of the abdomen too. Or the hydatid grows upwards into the thorax, causes dyspnœa and other pulmonary symptoms, and may eventually rupture into the left pleural cavity or bronchi. Examination of the blood often shows merely a slight degree of anæmia, and the leucocytes are normal both as regards their total number and their relative proportions.

Other Abdominal Echinococci. — (i.) *Peritoneum.*—The hydatids are usually multiple, possibly because of previous rupture or tapping of a cyst in the liver. (ii.) *Mesentery or omentum* is rarely affected alone. A single large cyst usually forms a swelling towards the middle line of the abdomen. There may be no symptoms till the cyst suddenly ruptures and causes peritonitis, or until the cyst suppurates, when there may be symptoms of acute intestinal obstruction as in a case of Stirling's, where the patient awoke one morning in great pain, and then for the first time noticed a swelling in the epigastrium. (iii.) *Pancreas.*—No definite case has been hitherto recorded. Subbotic reports the case of a patient, æt. 17, where the clinical features were those of a pancreatic cyst, but at the post-mortem it could not be ascertained whether the hydatid had developed from the pancreas or the mesocolon. (iv.) *Lumen of the Intestine.*—There is as yet only one case in the literature. In this case, one of multiple echinococci, Subbotic states that in the interior of the appendix there were three echinococci varying from a cherry-stone to a cherry in size, and also a degenerated echinococcus as large as a bean. The cysts had apparently caused no symptoms. (v.) *Primary Retroperitoneal Echinococci.*—The symptoms are very indefinite, and an accurate diagnosis before operation is almost impossible.

Karewski records two cases: (*a*) A woman, æt. 35, suffered for years from symptoms of lumbago without definite ascertainable cause until a swelling eventually appeared in the region of the left kidney. Operation revealed a suppurating hydatid which had developed behind the descending colon below the kidney. (*b*) A man, æt. 55, had suffered for many years from sciatica, and a large swelling formed with accompanying emaciation and fever. The condition was diagnosed as a retroperitoneal abscess, and was in fact a suppurating hydatid cyst.

Pelvic Echinococci are not common. In the female sex they are usually situated in the broad ligament, ovary, uterus, or pouch of Douglas, and an exact diagnosis can hardly ever be made unless the cyst rupture externally. A case affecting the Fallopian tubes is recorded in vol. iii. p. 253.

Echinococci in Bones.—Targett in 1894 stated that there were records of 76 cases. The echinococcus develops either in the medullary canal or in spongy bone. It is as a rule, but not invariably, the exogenous variety which is found. Brood-capsules and hooks are rarely seen, the echinococcus is not surrounded by a true fibrous capsule, and slowly infiltrates the bone. The medullary canal becomes distended, egg-shell crackling can sometimes be elicited, and the bone is very liable to be fractured. The condition is usually painless, the surrounding soft tissues are often invaded, so that at some part or other a cystic swelling approaches the surface, and the disease in the later stages is often complicated by periostitis and necrosis.

Stirling (1896) relates that a man, æt 43, had for five years noticed a slight swelling of the left thigh near the great trochanter. Since 1880 this thigh had been fractured by very slight violence five times. The patient was a healthy-looking man, well nourished, and had not suffered much pain, but the swelling had increased rapidly. The left thigh was found to be greatly enlarged in its whole extent, but especially at the upper part. The swelling was uneven and elastic in some places, especially at the inner and upper part of the thigh, where there seemed to be a distinctly cystic condition. Over the great trochanter there was a large, apparently solid tumour. There was no pulsatile movement and no lymphatic enlargement. The hydatid suppurated, the patient died, and at the post-mortem there was a large mother-cyst lining the cavity in the bone.

The symptoms of hydatid disease in the vertebræ may simulate those of Pott's disease; hydatid disease of the cranial bones produces a

bulging of the outer table, and may at the same time cause signs of cerebral tumour.

Echinococci of the Nervous System.—Sixty-eight cases of echinococci in the *brain* are recorded by Neisser. The cyst may develop in the meninges, cortex, or central white matter. It is usually single and of limited size, being as a rule as large as a nut or an orange. The adventitious capsule is either poorly developed or entirely awanting. The cyst grows slowly, and produces the general signs of a cerebral tumour, with localising symptoms according to its site. Sudden death may occur at any time from rupture of the cyst, but probably both the prognosis and the results of treatment are more favourable than in many other forms of cerebral tumour. Echinococci in the cerebellum are very rare, in the medulla and spinal nerves they are almost unknown. In the spinal canal about 14 cases have been recorded, and the cyst usually invades the dura mater from without. Blaschek finds that there are 59 cases of echinococci in the orbit on record; the cyst causes gradual exophthalmos and amblyopia, and a fluctuating swelling may sometimes be found between the orbital margin and bulb.

The heart is rarely affected. According to Roche there were in 1897 only 45 cases recorded, and in the case which he himself records there was sudden death, as so often occurs. Hydatid disease in arteries, veins, parotid, thyroid, muscles, subcutaneous tissues, and other sites is exceedingly rare. Gerulanos narrates a case where the muscles were affected with no less than 100 echinococcus cysts, varying in size from a lentil to that of two fists. A single echinococcus in the muscles is more common and may be mistaken for a lipoma, malignant tumour, or abscess.

Multiple Echinococci.—The symptoms vary according to the number, site, and size of the cysts. The liver is often enlarged, and there may be great abdominal distension, with fluctuating swellings in various parts of the abdomen, or more rarely in other parts, *e.g.* the axilla. Moore records such a case:—

The patient, æt. 38, had suffered occasionally from severe abdominal pains for a twelvemonth, and for the last seven months the abdomen had steadily increased in size. The patient had never any vomiting or jaundice. A number of rounded movable swellings could be felt all over the abdomen, but there was no ascites or hepatic enlargement. He was operated on four times, 47 cysts being dealt with in all, whilst at the first operation the whole omentum was seen to be studded with cysts varying in size from an orange to a hazel-nut. The patient made a good recovery.

Diagnosis.—The apparently good health and unimpaired nutrition of the patient, the history of the slow yet steady growth of the tumour, and the presence of a cystic swelling, are important. The diagnosis, however, must be mainly made by exclusion. The chief conditions from which hydatid disease of the liver has to be distinguished are distended gall-bladder, hepatic abscess, various hepatic tumours, subphrenic abscess, and pleuritic effusion. The initial site of the swelling should be ascertained. If this prove to have been, say, the right or left hypochondrium, pelvic hydatids can be excluded. If the swelling commenced towards the middle line, and is very movable, but does not move on respiration, the cyst has possibly developed in the omentum. If the swelling started in the left hypochondrium it may be impossible, until the abdomen is opened, to say whether the cyst belongs to the left lobe of the liver or the spleen. Echinococcus of the kidney develops behind the colon. Hydatid disease in the pelvis can in most instances only be suspected; the diagnosis must be made at the time of operation; similarly with cerebral and spinal echinococci. Multiple echinococci in the abdomen may be mistaken for tubercular peritonitis. The special points as to pulmonary echinococci are the copious sputum with its peculiar characteristic microscopic appearances, the localised area of dulness, and in most cases the absence, after repeated examination, of tubercle bacilli. A cyst in the lower lobe of the left lung may be indistinguishable from one of the spleen. To the Röntgen rays, all echinococci, not only those with calcified walls, are comparatively non-transparent, and as regards the size and extent of some abdominal echinococci, skiagraphy has been claimed to yield more information than ordinary physical examination. It may be useful, too, in locating pulmonary echinococci (Levy-Dorn and Zadek, *Berl. klin. Wochns.*, 1899).

If the cyst have ruptured, the diagnosis is usually comparatively easy. Otherwise a definite diagnosis is often impossible without an exploratory puncture. As fluid in some varieties of hydronephrosis and cerebro-spinal fluid closely resemble hydatid fluid chemically, the absence of albumin is not so characteristic of the latter as scolices, hooks, etc., the presence of which enables a positive diagnosis to be made. Tapping, however, unless it can be followed by immediate operation, should never be performed. For it is sometimes followed by serious symptoms, such as shock, syncope, dyspnœa, vomiting, convulsions, or high fever. Tapping is not infrequently followed by suppuration in the cyst, and the puncture of an abdominal hydatid may bring about the development of multiple echinococci of the peritoneum, or the trocar may wound some important structure such as the portal vein. An urticarial eruption, termed the "hydatid rash," may also appear after puncture, coming on rapidly, and involving the abdomen, thorax, face, and arms. The rash persists for 24 to 72 hours, and is usually accompanied by severe pruritus. These symptoms are thought to be

due to the absorption of some special toxic constituent of hydatid fluid. The toxic substance is capable of producing a great fall of the blood-pressure in the systemic arteries (Roy), and is regarded as a ptomaine (Mourson and Schlagendhauffen), or as a toxalbumin (Viron). The hydatid rash has been experimentally produced in the human subject by injection of hydatid fluid (Debove), but Graham found almost no disturbance produced by the injection of the fluid into dogs.

In the bones hydatid disease is most readily mistaken for sarcoma; but in the former there is a history of long-standing disease, the swelling is not pulsatile, and the lymphatic glands are not affected.

Prognosis.—Hydatid disease is always a serious condition because of the risk of suppuration or rupture of the cyst, the former being the danger of most frequent occurrence. The prognosis varies, however, according to the site and size of the cyst, on its degree of adhesion to neighbouring viscera, and on the ease with which it can be reached by the surgeon. Signs of degeneration and death of the parasite are favourable.

Prophylaxis.—In a district where the disease is endemic persons should be careful how they handle dogs, and if possible the number of dogs should be reduced to the necessary minimum. Dogs should not be allowed access to the water-supply, all drinking-water should be boiled, and uncooked vegetables thoroughly washed before being eaten. No dogs should ever be admitted to the slaughter-houses, and all offal containing hydatids should be destroyed, so that dogs may have no opportunity of eating it. The administration of anthelmintics to dogs has been recommended, as their use has been found of importance in reducing the frequency of "staggers" in sheep, whilst in Victoria one is advised to bury or throw boiling water over the exposed fæces of dogs, and to frequently purify with boiling water the kennels and the ground around them.

Treatment.—Although hydatids grow very slowly, and although many patients may undergo cure by spontaneous death of the parasite, yet every patient suffering from the disease should be treated, as his life may be at any time endangered by suppuration or rupture of the cyst. Surgical interference is the only mode of treatment in general use at the present time, and the methods of operation are as varied as are the characters of different hydatids. The procedure to be employed must therefore be that which appears best adapted to the individual case.

I. Incision of the cyst with drainage of the contents (marsupialisation of the French writers). A hydatid of the liver may be taken as an example. It may be dealt with (*a*) *by operation in one stage* (Lindemann). The abdominal wall is incised, the cyst exposed and brought up to the wound; swabs are applied to prevent any fluid passing into the peritoneum when the cyst is opened; the adventitious capsule is sutured to the edge of the parietal wound; the cyst is opened; the contents are thoroughly evacuated by irrigation, aided by gentle traction with forceps on the true cyst wall or daughter-cysts, and a large drainage-tube is inserted. In a favourable case healing will be complete in six to ten weeks. In Landau's modification the cyst is exposed, and is then tapped before the adventitious capsule is sutured to the parietes. (*b*) *Operation in two stages* (Volkmann). At the first operation the cyst is exposed and the wound packed with gauze. Adhesions will form between the serous surfaces, and about a week later the cyst is opened, emptied, and drained as in Lindemann's operation.

Indications.—Suppuration of the cyst, or any large cyst which from its size or configuration, or because it contains numerous daughter-bladders, cannot be completely and thoroughly emptied.

Disadvantages.—It is not uncommon for healing to be slow; a chronic fistula discharging bile may remain, the patient is exposed to the risk of septic infection, and finally a bad scar may result, with the possibility of subsequent ventral hernia.

If there are no peritoneal adhesions, which is rarely the case in suppurating hydatids, operation in two stages is claimed by some to be safer than Lindemann's operation, but in the former method the patient has to undergo a second operation, and the formation of adhesions cannot always be relied on even a fortnight after the first operation. There is in fact no advantage gained by operating in two stages, provided that in Lindemann's operation requisite precautions are taken to prevent the entrance of hydatid fluid into the peritoneum.

II. Evacuation of the cyst and closure of the abdominal wound. (*a*) *Modified Lindemann's Operation.*—After the cyst has been exposed and tapped, the adventitious capsule is firmly sutured to the edge of the parietal wound, the cyst is incised, the mother-cyst, daughter-cysts, and other contents are cleared out, the wall of the sac being thoroughly cleansed and dried with swabs. The edges of the incised adventitious capsule are then sutured together, and the parietal wound is closed. (*b*) *Intraperitoneal Treatment.*—The cyst is exposed, incised, thoroughly cleared out so as to remove all the mother-cyst, daughter-cysts, etc., and the sac is dried out with swabs. The edges of the incised adventitious capsule are sutured, the sac is dropped back into the abdomen, and the parietal wound is closed. There are various modifications of this operation. Delbet recommends the passing of sutures through the thickness of the walls of the sac so as to bring the latter in apposition. Posadas (*Rev. d. Chir.*, 1899, xix.),

however, not only regards Delbet's method as useless because the walls come naturally in contact without sutures, but as dangerous and impracticable. Tuffier scrapes the cyst wall and washes out the cavity with sublimate; others fill the cavity with iodoform glycerine.

In all these methods the wound is closed and no drainage employed. This is the procedure recommended of late by many writers besides those already mentioned. The procedure is applicable to non-suppurating cysts, to those where the whole of the mother-cyst and all daughter-cysts can be completely extirpated, and also, as stated by Posadas, to cysts which, by their adhesions to important viscera, or by their inclusion in such organs as are physiologically indispensable, cannot be completely removed by methods described below.

The healing of the wound by first intention obviates the intractable fistulæ, protracted convalescence, and other disadvantages of Lindemann's operation. One of the dangers of these methods, however, is that there may be recurrence of the disease owing to incomplete evacuation of the cyst; daughter-bladders, for example, may be left behind. Büdinger relates such a case. The liver was the seat of an echinococcus the size of a man's head, yet the patient had been operated on six years previously and regarded as cured. The special danger of the intraperitoneal method is that suppuration may occur in the sac and cause peritonitis, whereas should the sac suppurate, or its cavity fill up with bile after the "modified Lindemann's operation," the removal of a suture and a drain are all that is required (Stirling). Posadas records thirty-six cases treated by the intraperitoneal method. In twenty-eight cases there was healing by first intention; in three cases suppuration occurred, drainage was required, but the subsequent cure was rapid, and in five instances there was a fatal termination.

III. Enucleation may be performed in certain cases, *e.g.* a cyst in the liver or subcutaneous tissues, provided it be not larger than an ostrich's egg. The adventitious capsule is cautiously incised, and the echinococcus within is removed without a drop of fluid escaping (Posadas). Removal of the whole cyst *en masse* may be possible if the cyst be free, without adhesions, and with a pedicle, *e.g.* certain cysts of the omentum (Deguy); or extirpation of the cyst together with the organ—spleen, kidney, etc.—in which it has developed may sometimes be carried out.

Special points in the treatment of (*a*) *Subphrenic Hydatids.*—Access to the cyst has often to be obtained through the thoracic wall with resection of portions of one or more ribs. The pleural cavity will in many cases be obliterated at the level of the incision, but if not, the pleural surfaces are to be stitched together and the cyst fixed to the edge of the wound before the latter is opened. If irrigation and drainage are indicated a counter-opening below the costal margin is useful. (*b*) *Pulmonary Echinococci.*—Tapping is exceedingly dangerous because of the risk of drowning the patient with hydatid fluid. In one of Graham's cases the introduction of the needle led to the rupture of the cyst with consequent flooding of the healthy lung and sudden death. Operation is to be carried out on the lines indicated for subphrenic hydatids, except that as a rule no counter-opening is required. (*c*) *Echinococci of the Kidney.*—Most writers recommend incision with or without drainage. If the kidney appear to be entirely destroyed the advisability of nephrectomy may be considered. Sserapin states that of thirteen patients in whom nephrectomy was performed five died. (*d*) *Echinococci of the Spleen.*—The mortality of treatment by incision and drainage is stated by Roche to be 30 per cent. Splenectomy is facilitated by atrophy of the spleen and mobility of the tumour (Hahn), and Hartmann knew of eleven cases thus treated with only two deaths. (*e*) *Echinococci in Bones.*—Incision and scraping of the cyst with incision and drainage of abscesses may be tried, but suppuration is a serious occurrence, and if the disease be extensive amputation of the part is advisable.

Other methods of treatment must be mentioned, though they are now seldom if ever employed. I. Evacuation by aspiration and injection of (*a*) 25-30 c.c. of a 1-1000 solution of corrosive sublimate, which is left in (Hanot); (*b*) 100-500 c.c. of the same fluid, and evacuation of the same after leaving it in for ten minutes (Debove, Mesnard). Partial evacuation of the hydatid fluid (20-50 c.c.) and injection of a similar quantity of sublimate solution (Bacelli), or of a 1-5 per cent solution of silver nitrate (Berrucio). Iodine, carbolic acid, alcohol, etc., have also been used instead of sublimate. All of these methods are associated with the dangers of simple tapping.

II. Tapping was formerly the recognised method of treatment, but the associated dangers are so great that it should never be performed under any circumstances unless it immediately precedes operation by incision. In some instances the withdrawal of fluid does cause the death of the parasite, in other instances the fluid slowly reaccumulates, whilst finally in other cases the signs of refilling are simulated by the comparatively rapid growth of an echinococcus closely adjacent to the one which was tapped.

III. Electrical treatment bears with it the dangers consequent on tapping, yields unreliable results, and has never been generally adopted. Boinet, recording in 1897 a case treated by electro-puncture, is the only writer who has of late advocated the employment of electricity.

IV. Obsolete methods of historic interest. Such are the production of adhesions between the cyst and the parietes by the use of caustic pastes, or by the insertion into the cyst of two

trocars and cannulæ (Simon's method), with subsequent opening of the cyst; and, lastly, medicinal treatment, which has never proved to be of any real value.

Hydatid Mole. *See* Pregnancy, Pathology, Ovum and Decidua (*Affections of the Chorion*); Pregnancy, Hæmorrhage (*Hæmorrhage in First Three Months, Myxoma Chorii*); Puerperium, Pathology (*Sarcoma Deciduo-Cellulare*).

Hydatid of Morgagni. *See* Generation, Female Organs of (*Fallopian Tubes, Stalked Cysts*).

Hydatid Thrill.—The vibratory or trembling sensation given to the fingers of one hand during palpation over superficial hydatid cysts when the mass is at the same time percussed by the fingers of the other hand.

Hydracetin.—An antipyretic medicine, sometimes called pyrodine; acetyl phenyl hydrazine.

Hydradenitis Destruens Suppurativa. *See* Boils (*Symptoms, Axillary Boils*).

Hydræmia.—The morbid state in which the watery element of the blood is increased; it is practically a constant condition during pregnancy. *See* Pregnancy, Affections and Complications (*Blood Changes*).

Hydragogues.—Purgative drugs whose action is to cause a copious watery flow from the bowels; well-known examples are colocynth, gamboge, croton oil, jalap, and scammony. *See* Pharmacology.

Hydramnios or Hydramnion.—Excess of liquor amnii (above two pints at full term); hydrops amnii or polyhydramnios. *See* Achondroplasia (*Hydramnios as a Complication*); Labour, Diagnosis and Mechanism (*Transverse Lies, Causation*); Labour, Precipitate and Prolonged (*Faults in the Passenger, Liquor Amnii in Excess*); Pregnancy, Diagnosis (*Differential Diagnosis, Ovarian Cysts and Hydramnios*); Pregnancy, Pathology of, Ovum and Decidua (*Affections of the Amnion*); Pregnancy, Pathology of, Intra-uterine Diseases (*Diagnosis, Frequency of Hydramnios*).

Hydrargyriasis.—Mercurial poisoning or mercurialism. *See* Toxicology (*Irritants, Mercury*); Trades, Dangerous (*Mercury*).

Hydrargyrol.—The paraphenylthionate of mercury ($C_6H_4 . OHSO_3Hg$); has the form of brownish crystals; and has been used as a substitute for corrosive sublimate.

Hydrargyrum. *See* Mercury.

Hydrarthrosis.—Dropsy of a joint; effusion of serous fluid into an articulation. *See* Bursæ, Injuries and Diseases (*Bursitis, Chronic, Diagnosis*); Joints, Diseases of (*Definitions, Hydrarthrosis*).

Hydrastine and **Hydrastinine.** *See* Hydrastis; *also* Alkaloids.

Hydrastis Rhizoma.—The dried rhizome and rootlets of *Hydrastis canadensis*, sometimes known as Golden Seal. It contains two alkaloids—1. *Hydrastine*, in the form of white prismatic crystals, insoluble in water. *Dose*—½-1 gr. 2. *Berberine*. *Preparations*—1. Extractum Hydrastis Liquidum. *Dose*—5-15 m. 2. Tinctura Hydrastis. *Dose*—½-1ʒ. 3. Hydrastininæ Hydrochloridum, an oxidation product of hydrastine, in the form of pale yellow crystals, soluble in water; useful for hypodermic injection. *Dose*—½-1 gr. Hydrastis when applied locally has a stimulating action, and is employed as a lotion in the strength of 1 part of the liquid extract to 20 of water in the treatment of unhealthy ulcers, excessive local sweating, acne, and seborrhœa. An injection of the same strength may be used for leucorrhœa. A mixture of Hydrastinine and Protargol, each 1 per cent, with glycerine and water, is recommended as an injection for gonorrhœa, particularly in the later stages of the disease. Internally hydrastis is used empirically in all catarrhal conditions of mucous membranes and to increase contraction of the uterine muscle. It is given in chronic gastritis, particularly when following the abuse of alcohol. It is recommended also for catarrhal jaundice and catarrhal endometritis; for menorrhagia, dysmenorrhœa, and leucorrhœa. The tincture is said to have some action as an antiperiodic in malaria. "Liquor Sedans," a proprietary preparation largely used in dysmenorrhœa, is said to contain hydrastis.

Hydraulic Test.—The water test for drains; the lower end of the drain is plugged, then the drains are subjected to a pressure of a head of water of six feet for two hours, and it is observed whether there be any fall at the end of the time. *See* Sewage and Drainage (*Drains, Testing*).

Hydrazine.—An imaginary compound ($H_2N . NH_2$), occurring as hydrous hydrazine ($H_2N—NH_2, H_2O$), and forming salts with acids; phenylhydrazine ($C_6H_5HN—NH_2$) is used in the manufacture of colouring matters and of phenazone.

Hydrencephalocele.—A protrusion of brain substance, which contains in its interior a cystic cavity full of fluid, through an opening in the cranial vault.

Hydrencephaloid Condition.—A group of symptoms (semi-coma; contracted

and, later, dilated pupils; depressed anterior fontanelle; convulsions; squinting; retraction of the head) arising towards the close of life in cases of prolonged diarrhœa in children; spurious hydrocephalus.

Hydriatics.—The systematic treatment of disease with cold water (Gr. ὕδωρ, water, and ἰατρικός, healing); the water cure.

Hydroa. *See* Dermatitis Herpetiformis (*Synonyms*); Hæmatoporphyrinuria (*in Hydroa Æstivalis*).

Hydroadenitis. *See* Skin, Diseases of Sweat and Sebaceous Glands (*Sweat Glands, Suppuration in*).

Hydroarion.—Cyst of the Ovary. *See* Ovaries, Diseases of.

Hydrobromic Acid.—Acidum hydrobromicum dilutum (HBr) has been used, in doses of from 15 to 60 m., for the same purposes as the bromides, and also to prevent unpleasant symptoms arising during the prolonged administration of quinine; it is an official preparation.

Hydrocarbons.—Compounds of carbon and hydrogen, forming the paraffin, olefine, acetyline, anthracene, terpene, and benzene series, etc.

Hydrocele. *See* Scrotum and Testicle, Diseases of (*Testicle and Cord, Hydrocele, Congenital, Infantile, Funicular*, etc.). *See also* Fallopian Tubes (*Tubo-ovarian Cyst or Ovarian Hydrocele*); Fluids, Examination of Pathological (*Cystic Fluids, Hydrocele*); Hernia (*Inguinal, Diagnosis; Femoral, Diagnosis*); Neck, Region of (*Cysts, Acquired, Hydrocele of the Neck*).

Hydrocenosis.—The medical or surgical reduction of dropsy (by hydragogue purgatives or by aspiration).

Hydrocephalus.

See also Aspirator, Uses of (*Chronic Hydrocephalus*); Brain, Physiology of (*Venous Circulation, Pressure on Veins of Galen*); Brain, Hypertrophy (*Diagnosis from Hydrocephalus*); Brain, Surgery of (*Cephalocele*); Gastro-Intestinal Disorders of Infancy (*Chronic Diarrhœa, Spurious Hydrocephalus*); Head (*Shape*); Insanity, Pathology of (*Congenital Abnormalities*); Labour, Prolonged, Faults in the Passenger (*Congenital Hydrocephalus*); Mental Deficiency (*Classification, Hydrocephalic Cases, Morbid Anatomy*); Meninges of the Cerebrum (*Physiology, Causes of Hydrocephalus*); Orbit, Diseases of (*Malformations*); Syphilis (*in Children, Hereditary*).

Definition.—Hydrocephalus signifies a collection of fluid in the cavity of the cranium. The fluid may be situated between the dura mater and the brain, *i.e.* external or subdural hydrocephalus; or it may occupy and distend the ventricles, *i.e.* internal hydrocephalus.

Hydrocephalus may be acute or chronic, and the acute or chronic variety may be external or internal. The common form is chronic and internal, and is the only form which needs a detailed description here.

Acute Hydrocephalus.—This variety, whether external or internal, is the result of inflammation of the pia mater; it is doubtful if an acute hydrocephalus occurs under any other circumstances. Tuberculous meningitis is the chief cause; it is common to find at post-mortem examinations of those dying of this disease a large excess of fluid in the subdural space, and perhaps several ounces occupying the ventricles.

Chronic Hydrocephalus.—Chronic external hydrocephalus is a comparatively rare condition, and is usually secondary to some brain lesion. Whenever the brain, or a portion of the brain, undergoes shrinkage, a collection of fluid takes place occupying the subdural space corresponding to the atrophied part. A considerable amount of fluid may be present in some cases, as for instance when one-half of the brain has undergone partial atrophy. A collection of fluid may also be present in the subdural space in cases of chronic or pachymeningitis. Subdural fluid may be found in cases of internal hydrocephalus, in which the fluid has burst through some part of the brain and drained into the subdural space.

Chronic Internal Hydrocephalus.—In the common and important form of hydrocephalus the fluid is poured out into and gradually distends the lateral ventricles; as the fluid increases the head enlarges, the bones of the skull open out, the fontanelles bulge; there may be mental dulness and perhaps rigidity of the limbs.

Etiology. — Under normal conditions the amount of cerebro-spinal fluid in the ventricles and subarachnoid space is small. According to Leonard Hill "the living brain with its circulating fluid almost entirely fills the cranium, and the fluid that moistens its surfaces is little more in amount than the synovial fluid in a joint." It is true that, post-mortem, several ounces of fluid will drain away from the cranial cavity and spinal canal after the skull has been removed and the dura mater incised, but it is

probable that a certain amount of this is poured out during the act of dying, when the cerebral veins and sinuses are gorged and distended. During life under normal conditions any excess of cerebro-spinal fluid secreted in the lateral ventricles drains away *via* the third ventricle through the aqueduct of Sylvius into the fourth ventricle, and thence into the subarachnoid space through the several openings in the arachnoid membrane reflected from the cerebellum over the fourth ventricle (foramina of Majendie and Luschka). The cerebro-spinal fluid thus making its escape from the lateral ventricles into the subarachnoid space is absorbed by the veins. It is easy to understand if the "way out" of the lateral ventricles be occluded, fluid will be ponded up in the ventricles, and if the resistance to expansion be small, the amount of fluid will gradually increase and dilate the ventricles. A posterior basal meningitis is very liable to produce a matting of the structures in the neighbourhood of the fourth ventricle, and consequently occlusion of the foramina of Majendie and Luschka. The same result will follow if the aqueduct of Sylvius be blocked by a tumour, as pointed out by Hilton. Another mechanical form of chronic hydrocephalus is secondary to some obstruction of the veins of Galen by a tumour of the cerebellum or neighbourhood. The tentorium becomes stretched and the straight sinus compressed. As the veins of Galen drain the choroid plexuses and intra-ventricular veins, any obstruction to the former will lead to an excessive pouring out of fluid in the ventricles. It must be admitted, however, that the greater number of cases of chronic hydrocephalus, especially those which are congenital or which are first noticed during the first few months of life, are not the result of meningitis or tumour. In some of these cases a chronic inflammation (? syphilitic) of the choroid plexuses has apparently taken place, but in others no satisfactory explanation is forthcoming.

From the above considerations we may divide the cases of chronic hydrocephalus into two groups: the larger group includes the cases which in the present state of our knowledge we call *primary*, and which are either congenital or occur during the first months of life, and the secondary cases which follow a cerebral tumour or meningitis, and which may arise at any time of life and occur both in children and in adults. The symptoms in both groups, making allowance for age and the other lesions present, will be very much the same.

Symptoms.—The most marked and distinctive symptom of chronic hydrocephalus is progressive enlargement of the head. At birth the circumference of the normal head is roughly 14 in., at six months 16½ in., at a year old 18 in., at two years 20½ in. In well-marked cases of hydrocephalus the circumference may be increased by 2 or 3 in., and in extreme cases to as much as 8 or 10 in. In one of the author's cases the head measured 28 in. in an infant a year old. The shape of the head is globular, the cranium enlarging in the transverse more in proportion than in the longitudinal direction; the enlarged round cranium contrasts markedly with the face, the forehead and occiput strikingly overhang the face and neck. The fontanelles are widely open and bulge outward; they have a tense, elastic feel on pressure with the finger; the sutures are separated perhaps half an inch in some places. The scalp and skin over the forehead are stretched and shiny, and the superficial veins distended. The infant is unable to hold up its head, or indeed to have much control over its movements; it is usually mentally dull and takes but little notice of its friends, but this is by no means the rule, as it is often surprising to note that a hydrocephalic infant with an excessively large head will smile and show signs of moderate intelligence. In the worst cases the infant's health suffers, it gradually wastes, and its emaciated body contrasts greatly with the enormous size of its head; life is only prolonged a few months.

In other cases the course is more chronic, and the infant's health suffers less; it continues to live month after month, and in some cases year after year. It is unable to sit up or support its head, its intelligence is poor, though probably able to understand a good deal of what is said to it; the limbs become weak, especially the legs, the deep reflexes are exaggerated, with a tendency to spastic rigidity. In the most extreme cases which live to be a few years old, the eyes are pushed forwards and downwards, there is divergent squint, and difficulty in closing the eyelids. The limbs become flexed and rigid, there is complete amentia, and possibly atrophy of the optic nerves.

In a few cases in which hydrocephalus is not extreme, the head ceases to enlarge, and the skull may gradually close up. The child learns in time to walk and talk, but in the majority of cases the intellect never fully develops. Such children may be seen when a few years of age attending as the outpatients of a hospital, with large heads and uncertain gait; or they may be found in asylums as "hydrocephalic idiots"; or they drift into the workhouse as they are unable to earn their own living.

In a still smaller number of cases complete recovery appears to take place. The head ceases to enlarge, the fontanelles become less bulging, and in the course of many months or longer the head may regain its normal proportions. Improvement or recovery can hardly be expected in any but the slighter cases. Some of these cases are the subjects of congenital syphilis.

In secondary hydrocephalus the symptoms are likely to be mixed up with those of the

primary disease. Thus in the posterior basal meningitis of infants and children there will be the symptoms of meningitis, retraction of the head, etc., the fontanelle—if still open—will be tense and bulging, the skin of the forehead and scalp tense, and the circumference of the head increases in size. In these cases, if they survive, the enlargement of the head is seldom great; the hydrocephalus may perhaps be due to the obliteration of the foramina of Majendie and Luschka, or possibly to an intra-ventricular meningitis. When hydrocephalus occurs as the result of some obstruction to the veins of Galen, it is mostly at an age when the fontanelles have been closed and the sutures more or less firmly fixed than they are during infancy. In such cases the head will necessarily enlarge more slowly, and never reach extreme proportions, and in some cases there will be no enlargement at all. Perhaps the commonest cause of secondary hydrocephalus in children is a tubercular tumour of the cerebellum. As the tumour-growth proceeds the tentorium is stretched, the straight sinus and vena Galeni are obstructed, and fluid gradually accumulates in the lateral ventricles. In the more severe cases in children below the age of puberty the head may slowly enlarge, and the parietal and frontal bones separate, but this takes place probably in the later stages when the growth is of large size and the child bedridden. In the early stages, and in the slighter cases, when there is no enlargement of the head, the diagnosis of hydrocephalus is well-nigh impossible; in any case to differentiate between symptoms produced by the hydrocephalus and those produced by a tumour of the cerebellum is very difficult. A collection of fluid in the ventricles will be likely, by compressing the internal capsule and neighbouring parts, to produce paresis of the legs with exaggerated knee reflex. Some of the symptoms attributed to cerebellar tumour, such as uncertain and staggering gait, may be really due to the hydrocephalus which accompanies the tumour, and not immediately to the tumour itself. Hydrocephalus may be secondary to a sarcomatous tumour of the choroid plexus or blockage of the aqueduct of Sylvius by a tumour or clot of blood.

In certain cases in older children, in which the enlargement of the head is slight or not marked, chronic hydrocephalus has been found post-mortem, when death has occurred from some other cause. In such cases the course has been very chronic and suggestive of a slowly growing cerebral tumour, as there may be headache or more or less weakness of the limbs. On the other hand, vomiting and optic neuritis would be in favour of a tumour with or without hydrocephalus. Optic atrophy does, however, occur in some cases of chronic hydrocephalus apart from tumour. In these cases of chronic hydrocephalus the causation may be doubtful; possibly they may have suffered from congenital hydrocephalus and partially recovered, or they may be secondary to some slight basal meningitis.

In rare cases an escape of cerebro-spinal fluid from the nose, the fluid constantly draining away—much to the discomfort of the patient—has been practically the only symptom of chronic hydrocephalus.

Morbid Anatomy.—Normal cerebro-spinal fluid is clear and colourless as water, faintly alkaline, and of sp. gr. about 1005. It gives a slight opalescence with heat and acetic acid, and often a slight reduction of copper oxide when heated with Fehling's solution. The proteid present is globulin, not albumin, and the copper-reducing substance is pyrocatechin, not sugar (Halliburton). The fluid drawn off from many cases of hydrocephalus is identical with normal cerebro-spinal fluid; in other cases, as when it is the result of meningitis (posterior basal), the fluid is turpid and albuminous. The amount of albumin may be 0·3 per cent (Esbach) or more, and the specific gravity 1008. The amount of fluid present may vary from a few ounces to many pints. The changes in the brain consequent on a gradual distension of the ventricles will obviously be a compression and thinning of the brain substance which surrounds the ventricles. In extreme cases this is so great that the roof of the ventricles appears in places like a cyst with thin transparent walls. The fluid may burst through the corpus callosum or cortex. The floor of the ventricles is compressed and consequently flattened, and the internal capsule suffers compression. The aqueduct of Sylvius may be dilated and large enough to admit the little finger, while the floor of the 4th ventricle is also flattened by the pressure of the fluid. The choroid plexuses are usually normal in appearance, but a chronic inflammatory condition has been observed. The frontal, parietal, and occipital bones may be separated widely at the roof of the skull.

Diagnosis.—There can be no difficulty in making a diagnosis when the disease commences before the head has closed up. The gradual enlargement of the skull, the bulging fontanelles, and open sutures make recognition of the hydrocephalus easy. The most likely cases to present difficulty are those of rickety children, who, during their second or third year, have big heads with small bodies and limbs. The rickety skull is mostly flat on the top and square in outline, on account of the exaggerated frontal and parietal eminences; the superior fontanelle may be open, but not bulging, and the edges of the bones feel thickened. The sutures are closed; there are often shallow grooves corresponding to the transverse and longitudinal fissures. The brain in these cases is frequently larger than normal, but there is no hydrocephalus. Diagnosis is necessarily difficult

when the disease commences after the skull has closed up; gradual enlargement of the head with opening up of the sutures are the important points. Chronic hydrocephalus by itself, or in association with a cerebellar or other tumour, and without enlargement of the head, cannot be diagnosed with any certainty. Headache, paresis of limbs, exaggerated knee reflexes would probably be present, but it is needless to say these symptoms by themselves would not be distinctive.

Prognosis.—This is always grave, whether the disease is primary or secondary. In the majority of cases which arise within the first few months the disease steadily progresses, and death occurs within the first two years; in a few cases life is prolonged a few years, but rarely beyond the sixth or seventh. In a minority of cases the enlargement of the head ceases, the skull closes up, but the head remains abnormally large. In a very small number of cases complete recovery appears to take place. This can only be expected in the early stages, and when the hydrocephalus is slight or moderate in degree.

Complications.—Congenital cases of hydrocephalus may be associated with other and somewhat similar lesions as spina bifida, meningocele, and encephalocele. In some instances there has been club-foot or hare-lip.

Reference has already been made to a leakage of cerebro-spinal fluid from the cavity of the skull through the cribriform plate of the ethmoid bone into the nasal cavity.

Treatment.—In all cases of primary hydrocephalus, especially in those cases arising during the first few months of life, no time should be lost in commencing antisyphilitic treatment. The prospect of cure cannot be said to be very brilliant, but a certain proportion of these cases are syphilitic, and cures have been apparently effected by mercury and iodides, and it is at least worth while trying if they are of service, especially in early cases. Half a grain to a grain of grey powder may be given twice daily with one to two grains of iodide of potassium. This treatment should be persevered in for many months. Apart from such treatment it cannot be said that drugs are of much use, and medicinal treatment becomes a treatment of symptoms.

The Surgical Treatment of Hydrocephalus

Surgery has chiefly concerned itself with the treatment of chronic internal hydrocephalus or chronic distension of the lateral ventricles, and principally with those cases which are due to some cause other than cerebral tumour.

Occasionally need for surgical measures arises in cases which may be considered as examples of localised external hydrocephalus, where a collection of fluid is formed in some limited space either between the dura mater and the arachnoid or in the subarachnoid space so-called. Many of the instances of localised external hydrocephalus should rather be considered as examples of cysts resulting from blood extravasations or as passive collections of fluid poured out to replace an atrophied or shrunken brain. Though occasionally such conditions may require operative treatment, they hardly come within the scope of the present article.

Since the term acute hydrocephalus has been used as synonymous with acute meningitis due to tuberculosis or other conditions, the consideration of its treatment by operation must be briefly undertaken here, before the question of chronic internal hydrocephalus with which we are chiefly concerned is dealt with.

Acute Hydrocephalus.—If the evil effect of an acute meningitis, whether traumatic or simple or tubercular, was due entirely to pressure by a localised collection of thin fluid which could be readily drained away, its treatment by operation would be comparatively simple and probably satisfactory, the chief difficulty would arise in finding the position of the fluid, but it is obvious that other factors come into play, the fluid is only a part of the exudation, which may be mainly composed of lymph, and this by pressure upon important structures may render futile the attempt to give relief by drainage. Further, the inflammation may have involved the brain tissue and not merely the surface of the meninges, hence drainage in many cases cannot be expected to do more at best than give partial and temporary relief.

If, however, it is determined to try the effect of draining off the fluid, the attempt may be made either by trephining or puncturing the skull or by "lumbar puncture" (Quincke, *Centralblatt für Chirurg.*, 1892), *i.e.* tapping the spinal theca in the interspace between the third and fourth lumbar vertebræ, and thus either directly or indirectly the fluid effusion is drawn off. The question of draining for a longer or shorter period must be decided by the nature of the fluid; if this is sero-purulent or purulent it would, no doubt, be wiser to make provision for drainage: if the object was merely to relieve a suddenly increasing intracranial pressure, the removal of a certain quantity of fluid at the time would possibly be sufficient. If the puncture was made for the purpose of clearing up a doubt as to the tuberculous nature of the disease by examination of the fluid, aspiration would be sufficient, and might also produce a beneficial effect analogous to that obtained by removal of the fluid from a tuberculous peritoneal cavity.

For the performance of "lumbar puncture" it is only necessary to render the skin and syringe aseptic, and then with or without local or general anæsthesia to pass the needle between the laminæ of the third and fourth lumbar

spines slightly to one side of the middle line. The patient is made to stoop forwards, or the spine is fully flexed so as to widen the space as much as possible, and then the needle is passed gently in. If it enters to a sufficient depth and fluid is drawn off, it will be known that the theca has been tapped. If the needle impinges upon bone, its direction should be changed so that it passes below the edge of the lamina and enters the theca. After withdrawal of the needle the puncture may be sealed by a small collodium or celloidin dressing.

In a certain number of cases of meningitis, whether due to syphilis or tubercle or other cause, the ventricles become distended either with or without a subdural collection of fluid. Under these conditions the apertures leading from the ventricles to the sub-arachnoid space may be closed. A good many cases are on record where an operation has been done to drain away this intra-ventricular fluid as in chronic hydrocephalus. Thus Parkin (*Lancet*, 1st July 1893) trephined below the superior curved line of the occipital on the right side, opened the dura, and passed a bent probe beneath the cerebellum, thus setting free the fluid, which may be drained away either by a small tube, or better, by use of a bundle of horsehair. Parkin had a case in which a fatal result followed in sixteen hours. Ord and Waterhouse (*Lancet*, vol. i. 1894, p. 397) record a case of tuberculous meningitis, in which a very similar operation was successful, and the child, which was five years old, recovered after seventeen days' drainage.

A paper of Morton's (*Brit. Med. Jour.*, 8, 4, 1893) on the anatomy and method of reaching the intra-ventricular fluid without traversing the brain substance may also be consulted.

Chronic Internal Hydrocephalus. — The surgical treatment of this condition may be limited to an attempt to check or even diminish the rate of secretion of the fluid by pressure applied to the head. This has been done by strapping the head circularly with plaister, or by the application of an elastic bandage. Though such a simple and obvious measure has been many times attempted, it does not appear that any good result has followed, and there is real danger of causing sloughing of the scalp by compression of the vessels between the bandage and the unyielding skull. Another method of treatment is by tapping the ventricle through one of the lateral angles of the anterior fontanelle. The middle line is to be avoided on account of the presence of the superior longitudinal sinus. The tapping may be done with a fine hollow needle, a small trocar, or an aspirator, and after a certain amount of fluid has been drawn off, the opening may be sealed, or drainage for a time may be provided for by leaving in a capillary tube. For this purpose Southey's trocars have been employed. With this method of puncture may be combined the use of pressure to try to prevent reaccumulation of the fluid. Hern (*Brit. Med. Jour.*, 11th Nov. 1893) records six cases of repeated tapping, with improvement in five. A further step consists in the attempt to check intra-ventricular secretion by the injection of iodine or other irritating fluid.

Prof. Keen suggested making a trephine opening $1\frac{1}{4}$ inch above and behind the external auditory meatus, and then puncturing the brain with a needle directed towards a point $2\frac{1}{2}$ inches above the opposite meatus. The ventricular cavity was reached at a depth of about $1\frac{3}{4}$ inch. In a case in which this operation was done, a small horsehair drain was kept in the ventricle for fourteen days, and then a drainage-tube was substituted. Twenty-eight days later a similar operation was performed on the other side, and a tube passed into the ventricle. The child died on the forty-fifth day. Two other cases died four days and four hours respectively after operation. The first case was one of cerebellar tumour (*vide* Jacobson, *Operations of Surgery*, from whom the above is taken).

It is obvious that the measurements given above are only very vague guides, inasmuch as they appear to refer to adult heads or those in which the ventricles are not dilated. It is clear that in a dilated ventricle with a thinned brain the depth from the surface could be nothing like $1\frac{3}{4}$ inch. It should also be noted that in a case in which the hydrocephalus was a mere secondary effect of a cerebellar tumour no good result could be expected.

Mayo Robson (*Brit. Med. Jour.*, 1890, vol. ii.) trephined and punctured the ventricle in a case supposed to be one of basal meningitis resulting from ear disease. After failing to find pus he withdrew from the ventricle ℥vj. of clear fluid. The puncture was made in the posterior extremity of the second frontal convolution. No drainage was used. The child recovered. This case, again, can hardly be taken as a guide to the treatment of ordinary cases of chronic hydrocephalus. Mr. Robson points out that mere leakage or drainage away of cerebro-spinal fluid is not necessarily fatal, a fact well established by the records of cases of fractured skull, with laceration of brain involving the ventricle. He gives Prof. Fraser's directions for reaching the different cornua, though these are perhaps hardly necessary, since in any case likely to call for treatment a puncture through the cortex at any point of the upper part of the hemisphere would reach the dilated ventricle. Care will, of course, be taken to avoid the motor area. Robson records another case of ordinary hydrocephalus in which he operated, but the drainage-tube slipped out and the child died.

Bruce Clarke, in the discussion on Robson's paper, mentions a case of chronic meningitis with subdural collection of fluid (external

hydrocephalus) which he drained. The patient died in four days. Kendal Franks tapped a ventricle with temporary improvement, but death occurred in four days, which seems about the usual time. In this case the ventricle, after death, contained but little fluid, but there was a purulent basal tubercular meningitis. Wheelhouse had a similar experience.

Jacobson, to whom we owe several of these references, quotes a case of Broca's, in which drainage appears to have been successful up to the fiftieth day, when the wound was healed and the child discharged.

The operation of Parkin already described was performed on a child of eleven months with chronic hydrocephalus. The child was becoming comatose, but after eighteen days' drainage recovered and became more intelligent than it had been before the onset of the acute symptoms. The plan would apparently be applicable to cases in which the foramen of Majendie was closde by adhesions.

Sutherland and Watson Cheyne (*Clin. Soc. Transactions*, vol. xxxi.) devised and carried out a plan of draining the ventricles into the subdural space. An opening was made in the left lateral angle of the anterior fontanelle (the child was six months old), and a bundle of fine catgut tied together at one end was passed into the subdural space, the other end of the bundle was by means of sinus forceps carried into the lateral ventricle, and the wound closed. The size of the head diminished steadily, and for a time the child did well, but a convulsion came on after the first month, and after nine weeks it began to go downhill, and died three months after operation, with rapid wasting, screaming, and opisthotonos. The shrinkage in size of the head was remarkable, and good drainage had been apparently established. The principle of the operation in establishing drainage and preventing intraventricular pressure is good, and the operation is apparently very simple. The length of drain should be only sufficient to project $\frac{1}{4}$ inch into the ventricle. The child was syphilitic, but the plan, though not ultimately successful, is more promising than the other methods devised, except, perhaps, that of Parkin. In this case, as in so many other operations, craniectomy, simple tapping, and so on, the temperature rose high at first, nearly to 106°, but subsided gradually, and was clearly not due to septicity.

Starr (*Brain Surgery*) records some other cases of operation for hydrocephalus. In 1898, Bruce and Stiles (*Brit. Med. Jour.*, 29th Jan. 1898) treated a congenital syphilitic child of thirteen years suffering from basal meningitis and severe and increasing symptoms of intracranial pressure by an operation like that of Parkin's, except that the opening was made in the middle line, and included the margin of the foramen magnum. On separating the amygdala a large quantity of fluid escaped and continued to drain away. The child improved, but died after fifteen days of hyperpyrexia, possibly the result of renal disease.

On the whole it is probably best, in any case with progressive symptoms, to try either Cheyne's or Parkin's method.

Hydrochloric Acid. *See also* Digestion and Metabolism (*Gastric, Secretion*); Indigestion (*Acidity*); Œsophagus (*Inflammation, Causes*); Physiology, Food and Digestion (*Gastric*); Stomach and Duodenum, Diseases of (*Gastritis*); Toxicology (*Inorganic Poisons, Corrosives*). Acidum Hydrochloricum. HCl. A clear colourless fuming liquid, obtained by dissolving in water the fumes produced by the action of sulphuric acid on sodium chloride. Contains 31·79 per cent of hydrogen chloride. *Preparations* — 1. Acidum Hydrochloricum Dilutum (contains 10·58 per cent of hydrogen chloride). *Dose*—5-20 m. 2. Acidum Nitrohydrochloricum Dilutum. Nitric acid, 3; hydrochloric acid, 4; distilled water, 25. *Dose*—5-20 m.

Pure hydrochloric acid has a caustic action when applied to skin or mucous membrane, but as it is the weakest of the mineral acids it is hardly ever employed as an escharotic. In very dilute solutions it may be used as a refrigerant lotion in febrile conditions. Internally the dilute forms are prescribed for their action on primary digestion. They stimulate the flow of saliva and are also said to increase the amount of bile. Their effect on the secretion of hydrochloric acid in the stomach depends on whether they are given before or after a meal. When the stomach is empty they are said to diminish the formation of hydrochloric acid and are therefore indicated in dyspepsias associated with acid eructations and heartburn. Given after a meal they simply augment the acidity of the gastric contents and are given where dyspepsia is due to deficient secretion. Small doses are recommended in typhoid and other febrile conditions, the natural secretion of acid being defective when the temperature is raised.

Hydrocirsocele.—A combination of hydrocele with varicocele.

Hydrocyanic Acid. *See also* Breath (*Odour of*); Toxicology (*Cyanogen Compounds*). Prussic Acid. *Acidum Hydrocyanicum Dilutum*, containing 2 per cent by weight of hydrogen cyanide, is the official form. It is a colourless, volatile, unstable liquid with an almond-like odour. *Dose*—2-6 m. *Preparation*—Tinctura Chloroformi et Morphinæ Composita, an imitation of chlorodyne, each 10 m. containing $\frac{1}{2}$ m. of dilute hydrocyanic acid. *Dose*—5-15 m. Hydrocyanic Acid is also contained in Aqua Laurocerasi and in oil of bitter almonds.

Hydrocyanic acid is useful locally as a sedative

to allay itching in such conditions as pruritus ani or vulvæ, for which purposes a 2 or 3 per cent solution will be found sufficient. It must not be applied if the skin is at all broken, as there is danger of poisoning from absorption. Internally it is given to relieve gastric pain and vomiting, especially when these are of purely nervous origin. It is frequently of great value in the treatment of irritable tickling cough, either alone or as an ingredient of a cough mixture.

Hydrocystoma. *See* Skin, Diseases of Sweat and Sebaceous Glands (*Sweat Glands, Hydrocystoma*).

Hydroformant Lamp.—A means of disinfection by fumigation, in which paraformaldehyde tablets are volatilised by the steam from boiling water in the lamp.

Hydrogen Peroxide.—H_2O_2. Is used in medicine as an aqueous solution, *Liquor Hydrogenii Peroxidi*, which should yield ten times its volume of oxygen. It is a colourless, odourless liquid with a slightly acid taste. *Dose*—½-2ʒ.

It has been recommended for the treatment of diabetes, epilepsy, uræmia, dyspepsia, and many other diseases; but there are no proofs of its efficacy in such conditions, and it probably is of no value in internal medication. For external use its action depends entirely on the oxygen given off when it comes in contact with any purulent material. It is used in ulcerative stomatitis, in tonsillitis, and in diphtheria. It is also a valuable preparation for cleansing ulcers and malignant growths. It is an excellent disinfectant and deodorant for injection into foul abscess cavities, but when used for this purpose care must be exercised that there is a free exit for the effervescing material that is given off. A plaster-of-Paris casing is softened and cuts easily after the application of peroxide of hydrogen, but acetic acid is equally good for this purpose and much less expensive. The pain accompanying the removal of strips of adhesive plaster is lessened by the application of peroxide. It has also been said to relieve the pain caused by wasps' stings. It is the active ingredient of the disinfectant fluid known as "Sanitas."

Hydrogen Test. *See* Abdomen, Injuries of (*Diagnosis of Intestinal Perforation*); Bladder, Injuries of (*Rupture, Diagnosis of*).

Hydrometra.—The distension of the uterine cavity with a watery fluid.

Hydromyelia.—Dilatation of the central canal of the spinal cord with fluid; sometimes used as a synonym of syringomyelia and even of spina bifida. *See* Spinal Cord, Medical (*Anatomical Considerations, Central Canal*); Spinal Cord, Medical (*Spina Bifida*); Syringomyelia (*Pathological Anatomy*).

Hydromyringa or **Hydromyrinx.**—Dropsy of the tympanum. *See* Ear, Examination of (*Tympanum, Fluid in*).

Hydronephrosis. *See* Abdominal Aneurysm (*Pressure Symptoms*); Ascites (*Diagnosis*); Circumcision (*Phymosis, Complications*); Kidney, Surgical Affections of (*Hydronephrosis*); Prostate Gland (*Pressure Symptoms*); Urethra, Diseases of (*Stricture, Sequelæ*); Uterus, Non-Malignant Tumours of (*Fibroids, Pressure Results*).

Hydropathy.

See also Balneology (*Action and Kinds of Baths*); Mineral Waters (*Baths*).

Hydropathy, or treatment by water, is a most patent method either for good or ill. It covers an enormous amount of ground; the uses of water—hot, cold, and frozen—internally and externally, by the aid of various mechanical methods, and in both acute and chronic disease. The drinking of water is absolutely necessary; without it life would be impossible. The human body contains about 59 per cent of water; there is a constant loss going on through the agency of respiration and the various secretions which must be made good. The internal functions of tissue change and the expulsion of waste products depend largely upon water.

The drinking of cold water lowers for the time the temperature as taken in the axilla or rectum, reduces the frequency of the pulse, but increases its tension. These effects vary according to the quantity of water taken and the rapidity with which it is swallowed; the greater the quantity and the more quickly it is drunk the greater the result.

The effect upon the pulse-rate is probably due to stimulation of the terminal branches of the vagus in the stomach, upon the tendency to stimulation of the vaso-motor centre.

When drunk warm it causes diminished tension and a slight increase of pulse-rate, due, it has been thought, to nausea, causing a lowering of tone in the nerve-centres.

Hot-water drinking (110° to 115° F.) seems to exert much the same influence on pulse-rate and tension as cold water, but slightly raises the general temperature of the body.

The drinking of hot water is indicated when

it is desired to stimulate the stomach to increased action and to wash out the system generally. It is more diffusible than cold water, and does not depress either by lowering temperature or by using vital force for the purpose of reheating the organism.

Cold-water drinking sometimes stimulates the lower portions of the alimentary canal. Many invalids, with failure of digestive power, find that warm water taken with or just after food is less depressing than cold water.

For the purpose of washing out the system, aiding the removal of tissue waste and the escape of the various toxines, water should be taken in the early morning, the next best time being one hour before the mid-day or evening meals. Some gouty patients find a tumblerful of hot water at bed-time of great service.

The dosage of water varies. Women, who as a rule take too little fluid, except with food, have to commence with from four to six ounces. The subjects of gastric atony have also to take small doses at first. In other cases, especially in the gouty, a pint of hot water may be taken every morning fasting, the drinking being spread over ten or fifteen minutes. Nausea is sometimes produced by drinking hot water. This may be got over by making the water hotter, or by adding a dessertspoonful of fresh lemon juice to each pint. This addition as a rule suits the gouty and dyspeptic—recent researches showing that it checks the formation of duodenal ferments and toxines.

It has been alleged that the drinking of large quantities of water dilutes the blood unduly, but expert evidence is against this view, and shows that part of the excess of water quickly passes though the kidneys, the rest going to dilute the various fluids in the tissues. It is probable, however, that in marked gastric atony the fluid does not leave the stomach as quickly as it should, and so aggravates the morbid condition. Water-drinking causes marked diuresis, and urea, chlorides and phosphates are passed in increased quantities; the flow of the saliva, pancreatic fluid, and bile is augmented.

External Uses of Water.—The physiological effects are very fully treated of in the article "Balneology," vol. i. p. 341, and the special effects of baths given in water containing various salts and gases are also dealt with in the same article.

The hot bath, 96° to 110°, causes an increased flow of blood to the skin, the pulse is quickened, as is also the respiration. The skin becomes red and perspiration follows.

The tepid bath, 84° to 94°, has a calmative effect, and does not excite either the nervous system or the circulation.

The cold bath, 50° to 60°, at first causes loss of superficial temperature, increased capillary contraction, and an action on the central nervous system through its peripheral branches. Then reaction takes place, followed by redness of skin, increase of temperature, and a certain amount of nervous excitement.

The effects of all these baths depend upon the length of immersion.

Average duration:—hot bath, 8 to 10 minutes; tepid bath, 10 to 60 minutes; cold bath, 3 to 10 minutes.

Longer immersions in either hot or cold baths are often followed by marked depression.

Hydropathic baths and methods consist of thermal and mechanical measures which stimulate the whole body or parts thereof. These give rise to—

(1) Raising or lowering of temperature.
(2) Improved circulation through the peripheral vessels, lymph spaces and channels, and increased elimination of toxines.
(3) More efficient oxidation.
(4) Improvement in general vascular and nervous tone.

Hydropathic Methods.—The chief measures employed are the following:—

The Shower Bath.—Temp. 100° to 60° or 50°. Duration, one to two minutes.

The Needlebath or *douche en cercle.*—This consists of two curved horizontal pipes connected by a number of smaller vertical pipes. These vertical pipes are perforated with small apertures at intervals of an inch and a half, the result being that many forcible jets of water impinge simultaneously upon practically the whole body surface. Temp. 100° to 60° or 50°. Duration, one to three minutes.

Douches.—(*a*) *Ascending.*—The patient sitting on a seat, like that of a water-closet, water is delivered vertically upwards through a ¾-inch nozzle, or through a small rose, so as to impinge upon or even enter the fundament. Temp. 100° to 60° or 50°. Duration, half to two minutes.

(*b*) *Descending.*—A stream of water from a 1½-inch pipe is poured from above. The patient stands under this, moving slightly backwards and forwards to allow the water to pass down back and front of body. Temperature and duration as last bath.

(*c*) *Spinal.*—A stream of water directed from 1½-inch pipe and played up and down the spine. Temperature and duration as last.

(*d*) *Scottish.*—Alternate jets of water at temperature 100° and 50°, played upon the spine, changing six times in the minute. Duration, half to two minutes.

(*e*) *Aix Douche.*—A combination of hot douche with massage. The patient sits on a short-backed wooden chair, the feet being covered with hot water, and a fixed spray at 98° plays upon the back and shoulders. Meanwhile the masseur, directing a stream of water from an open hose-pipe upon the part, rapidly manipulates the front of the body and afterwards the back. The proceeding may be varied by a

second attendant simultaneously treating the back. At the end of the bath the patient stands at the extreme end of the room grasping a bar, and a strong pressure douche is played over the body in the direction of the lymph stream, following the course of the lymphatic vessels. A valuable modification, very useful in many cases, is to let the patient lie in a shallow elevated bath, and the attendant giving the massage under the douche, finishing with the final douching as mentioned above.

(*f*) *Underwater Douche*.—An invaluable aid to treatment, especially that by natural mineral water baths. While the patient is sitting in the bath a pipe with a ¾-inch nozzle is placed in the bath, and water under pressure at a temperature of from 96° to 110° is applied to the affected parts, the force being graduated by keeping a cushion of water between the end of the nozzle and the part douched.

Packs.—(*a*) *Hot*—general or local. A thick blanket is folded and rolled up in bottle form. Boiling water is poured through the two ends in turn: it is covered with a large hot towel, and as much superfluous moisture as possible wrung out. It is then spread out on the top of two large dry blankets spread on the bed; on a spare one, if the patient is confined to bed. The patient, divested of clothing, is placed on the hot wet blanket and rapidly covered up by this and the dry ones. Hot water tins should be placed outside the blankets and to the feet, and cold water cloths to the head. The operation should be done quickly, so that the wet blanket does not lose heat. Duration, 30 to 60 minutes.

(*b*) *Cold*.—Two large thick towels are wrung out of tepid or cold water. The patient is laid on one and covered by the other, and a single blanket is lightly placed over all. Every ten or fifteen minutes the upper towel should be replaced by one wrung out of cold water; the lower one may be changed every hour, if required. Duration, from 30 minutes to two hours.

(*c*) *Mustard*.—Mustard bran is mixed into a thin paste with boiling water. It is spread on muslin rag and covered by a piece of the same. It is then placed over the affected part and kept on for 10 to 20 minutes, covered by a flannel bandage.

Compress: hot or cold.—A piece of thick Turkish towelling is wrung out of hot or cold water and applied to the affected part. It is then covered by flannel, outside which a piece of Mackintosh sheeting is applied. Duration, 20 to 30 minutes.

Hot-Air Applications.—(*a*) *Turkish Bath*.—Fully described in article "Balneology," vol. i. p. 349. Temperature, 115° to 200°. Duration, 20 to 60 minutes.

(*b*) *The Hot-Air Box*.—The patient sits in this with head protruding, the feet being placed in hot water and a cold towel applied to the head. Temperature, 108° to 120°. Duration, 8 to 12 minutes.

(*c*) Hot-air bath given in bed, patient covered by wicker cage; lamp out of bed and heat conducted through metal tube. Duration, 15 minutes to two hours or more.

(*d*) Local hot dry air applications:—

(1) In box as before described through holes cut for insertion of hands or feet.

(2) "Greville" Method.—The limbs placed in cylinders heated by electricity. Temperature, 180° to 450°, or as much as can be borne. Duration, 30 to 80 minutes.

(3) "Dowsing" Method.—The electric lamp bath with reflectors. Temperature and duration as above.

(4) "Tallerman" Method.—The limb placed in a copper cylinder heated by a large number of gas jets. Temperature, 110° to 250° or more. Duration as above.

The whole body as well as individual limbs may be treated by the "Greville" and "Dowsing" methods.

Vapour Applications:—

(*a*) Russian Bath.—A chamber heated by steam to a temperature of 110° to 120°. Duration, 10 to 20 minutes.

(*b*) Vapour Box Bath.—Temperature, 108° to 120°. Duration, 8 to 15 minutes.

(*c*) Local Vapour Baths.—Hands or feet placed in box through holes cut for purpose. Temperature, 110° to 120°. Duration, 5 to 20 minutes.

Sitz-baths.—Patient sits in special bath, the water just covering the groin. The back and shoulders should be covered by a blanket, and the feet should also be wrapped up. May be given hot, tepid, or cold. Duration, 2 to 10 minutes.

Hydropathy is of service in both acute and chronic disease.

Acute Diseases.—*Acute Rheumatism*.—A hot blanket pack, as before described, is often most useful. May be repeated twice daily if required.

Rheumatic Hyperpyrexia.—These cases are so quickly fatal that rapid and energetic methods are called for. The writer is strongly in favour of the *immediate* use of the cold bath, and believes that in many cases the time wasted in cold sponging or cold packing has determined a fatal issue.

Whenever the temperature in acute rheumatism touches 105° the patient should be carefully lifted in a sheet by five persons—one at each corner, the fifth supporting the head. A bath should be placed close to the bed, and should contain from 18 inches to 2 feet of water at 92°. This should be cooled down by the addition of ice or cold water to 72°, and the patient kept in till the temperature falls below 100°. Ice should be applied to the head during the bath.

Careful attention must be paid to the condition of the heart, which will, of course, govern the duration of the bath : this is usually 10 to 30 minutes. The patient must be carefully lifted in the sheet and replaced in bed on a hot blanket, with a hot water bottle to the feet. After a short time both sheet and blanket may be drawn away.

The bath should be repeated as often as the temperature reaches 105°.

The writer has known as many as eight baths given in twenty-four hours with recovery of patient.

Enteric Fever.—Various opinions are held by competent authorities as to the respective value of cold baths, cooling packs, cooling towels, and cold sponging in this disease; but nearly all are in agreement as to the benefit derived from one or other of these methods.

The advocates of the routine bath treatment say that whenever the temperature rises above 103° the patient should be immersed in a bath at 90°, cooled down to 70° or even 60°. Period of immersion, 10 to 20 minutes; the method of administration being as before described. The objection to this treatment is that it needs to be given with the greatest skill and care, as, when bowel complications are present, much damage may be done by injudicious movement, and preference is often given to the following methods :—

Cold pack, as before described. This method may be continued until the temperature falls below 100°.

Cooling Towels.—Pieces of thick Turkish towelling, 2 feet by 1 foot, are wrung out of tepid water and applied from the top of the chest to lower part of abdomen, across the front of the body, and a piece of flannel placed over them: every few minutes the towels may be wrung out of cold or iced water and changed. This process does not necessitate any movement of the patient, and can be applied continuously.

Sponging with cold or iced water may be done every hour, the various parts of the body being uncovered, sponged, and covered in turn.

Ice packing is sometimes resorted to in extreme cases, but as a rule is not so convenient nor so efficacious as the above methods.

During these applications of cold to the body the head should be kept cool by an ice cap or cold-water bag.

Scarlet Fever.—In those cases of scarlet fever where the rash fails to come out and the patient is threatened with death from hyperpyrexia, the treatment is exactly the same as for rheumatic hyperpyrexia.

The bath and not the milder cooling methods is the only remedy.

Typhus Fever.— Here the cooling pack is beneficial.

Pneumonia.—In the pneumonia and broncho-pneumonia of children the cooling towel is most useful, and should be used whenever the temperature rises above 102°.

In adults the application of cold has to be made more cautiously, but whenever the heart seems to be failing from high temperature the cooling pack or cooling towels should be used; and in extreme cases with very high temperature the cold bath, but the latter should be used with the greatest care. Large enemata of cold water reduce temperature and are sometimes useful.

Uræmia.— The hot blanket pack, before described, is sometimes useful here, but not nearly so much so as the hot-air or vapour bath given in bed. The bedclothes being supported by cages, a large spirit lamp is placed on the floor at the foot of the bedstead, and the hot air or vapour is conducted into the bed by means of a tube. The bath may be kept going for from fifteen minutes to two hours, and may be repeated as often as required. It has frequently succeeded when all other methods have failed, especially in nephritis following scarlet fever, and in some cases of puerperal convulsions.

Acute gout is often at once relieved by local hot-air or vapour baths.

Subacute and Chronic Diseases.—*Rheumatism and gout* are benefited by the use of the hot-air and vapour baths, general or local. In cases of gout the vapour generally seems more useful, and is an invaluable adjunct to treatment by natural mineral water baths.

Lumbago, myalgia, sciatica, neuralgia, and neuritis are also efficiently treated by these methods, with the help of the hot underwater douche; also by the Aix douche. The more chronic forms of sciatica are relieved by the Scottish douche.

Rheumatoid arthritis, if of the rheumatic or gouty type, is relieved by the local application of hot dry air, especially in conjunction with the constant current or sinusoidal or electric bath; in the more purely neural forms the hot air is of limited service, and should be used with great care, the electric bath being much the most effective treatment.

Neuritis is often benefited by the application of mustard packs over the painful nerves, especially over tender spinal points, and they are often given in connection with the sinusoidal electric baths which have done so much for neuritis.

Affections of the skin, as dry eczema, psoriasis, lichen planus, etc., are well treated by hot dry air; the more inflammatory condition by the steam box bath or local application of vapour.

Chronic Bright's disease, renal insufficiency, suppressed gout, lithæmia, and other forms of auto-intoxication, can be safely and well treated by the vapour or hot-air baths.

Neurasthenia.—Needle bath, Scottish douche,

mustard pack over the cervical ganglia of the sympathetic.

Dyspepsia and Hepatic Congestion.—Needle bath, Scottish douche, mustard pack over cervical ganglia of sympathetic and over liver and stomach.

Piles.—Ascending douche.

Leucorrhœa, dysmenorrhœa, and other uterine troubles, sitz-bath with ascending douche.

Obesity.—Needle bath, spinal douche, Scottish douche, Aix douche.

Constipation.—Scottish douche, cold spinal douche, large enemata of water to wash out colon.

Ice.—May be used internally or externally. It is useful sucked, or swallowed in small pieces, in inflammatory conditions of, or hæmorrhage from mouth, tonsils, throat, or stomach, and in vomiting it also lowers temperature and sometimes stops hiccough; placed in the rectum it lowers temperature.

Externally it has many uses. It is applied to the head in inflammatory and feverish affections: to the neck in acute tonsillitis. Application to the spine and over the epigastrium relieves hiccough, and is often placed over lungs and stomach in hæmorrhage from those organs.

It has been used with much success in the treatment of pericarditis and endocarditis, applied for short periods over the heart; and tachycardia has often been relieved by this application. It relieves acute inflammatory affections of joints, and also some cases of neuritis. Certain obstinate cases of acute or subacute rheumatoid arthritis, especially of the knee, have done well with ice massage, twice daily, for half an hour each time. A flat piece of ice is taken in the hand of the attendant, and gentle rubbing is given. The application of the ice bag to the lower spine is said to stimulate the utero-ovarian organs; while applied over the lower part of the abdomen it stops uterine hæmorrhage; it is sometimes serviceable in the early stage of strangulated hernia, enabling reduction to be made without operation.

Hydropericardium.—The presence of serous fluid (non-inflammatory in origin) in the pericardial sac. See Pericardium, Diseases of (*Hydropericardium*).

Hydroperione.—Liquid lying between the decidua vera and the decidua reflexa; it has been regarded as serving for nourishment to the embryo.

Hydroperitoneum.—The accumulation of serous fluid in the peritoneal cavity. See Ascites.

Hydrophidæ. See Snake-Bites (*Colubrine, Hydrophidæ or Sea Snakes*).

Hydrophobia. See Rabies; Hysteria (*Varieties of Attacks, Hydrophobic Phenomena*); Tetanus (*Diagnosis*).

Hydrophthalmos. See Cornea (*Changes in Hydrophthalmos*); Glaucoma; Sclerotic, Diseases of (*Staphyloma*).

Hydropneumopericardium.—The presence of air and fluid in the pericardial sac.

Hydropneumothorax.—The presence of air and fluid in the pleural cavity. See Pleura, Diseases of (*Pneumothorax*).

Hydrops.—Dropsy. See Ascites; Fallopian Tubes (*Hydrops Tubæ Profluens*); Joints, Diseases of (*Definitions, Hydrops*); Joints, Diseases of (*Tuberculous, Hydrops Tuberculosis*); Joints, Diseases of (*Arthritis Deformans, Hydrops*); Knee-Joint, Diseases of (*Tuberculous, Hydrops Tuberculosis*); Labour, Prolonged, Faults in the Passenger (*Hydrops Amnii*); Lacrimal Apparatus, Diseases of (*Lacrimal Sac, Hydrops*); Pregnancy, Pathology of, Ovum and Decidua (*Affections of Amnion, Hydrops Amnii*).

Hydrops ad Matulam. See Diabetes Mellitus.

Hydroquinone.—An isomer of resorcin and pyrocatechin ($C_6H_4 . O_2H_2$); it is an oxidation product of phenol (paradihydroxybenzene); the difference in properties existing between the three isomeric substances (resorcin, pyrocatechin, and hydroquinone) is supposed to be due to the relative positions of the two hydroxyl groups in the molecules; as an antiseptic hydroquinone has been used in typhoid fever, rheumatic fever, etc.; it may be found in the urine. See Toxicology (*Corrosives, Carbolic Acid, Symptoms*); Urine, Pathological Changes in (*Colour in Carboluria, Ethereal Sulphates*).

Hydrorrhachis.—Spina bifida (*q.v.*).

Hydrorrhœa.—A watery discharge, especially that which takes place, intermittently, from the pregnant or less often from the non-pregnant uterus (*hydrorrhœa gravidarum*); hydrorrhœa may also occur in nasal disease. See Nose, Nasal Neuroses (*Hydrorrhœa or coryza vaso-motoria periodica*); Nose, Foreign Bodies, etc. (*Nasal Hydrorrhœa*); Pregnancy, Pathology of, Ovum and Decidua (*Affections of Decidua, Inflammation, Hydrorrhœa Gravidarum*).

Hydrosalpinx.—A collection of watery fluid in the Fallopian tube, causing it to assume a retort or sausage shape. See Fallopian Tubes (*Hydrosalpinx or Dropsy of the Tube*); Menstruation and its Disorders (*Dysmenorrhœa, Causes, Tubal Disease*).

Hydrostatic Test. *See* Medicine, Forensic (*Infanticide, Proof of Live Birth*).

Hydrotherapy. *See* Balneology; Hydropathy; Hysteria (*Treatment*); Mineral Waters; Tabes Dorsalis (*Treatment, Hydrotherapy*).

Hydrothionæmia.—The presence of sulphuretted hydrogen or sulphide of ammonium in the blood, due to perforation of the bowel, etc., and causing serious toxic symptoms (vertigo, collapse, etc.). *See* Unconsciousness (*Auto-intoxications*).

Hydrothionuria. — The presence of sulphuretted hydrogen in the urine from bacterial decomposition. *See* Urine, Pathological Changes in (*Sulphur, Hydrothionuria*); Urine, Bacteria in (*Hydrothionuria*).

Hydrothorax.—The presence of serous fluid (non-inflammatory) in one or both pleural cavities. *See* Heart, Affections of Myocardium and Endocardium (*Symptomatology, Dropsy*); Labour, Prolonged, Faults in the Passenger (*Diseases of the Child, Hydrothorax*); Pleura, Diseases of (*Hydrothorax*).

Hydrotympanum.—Presence of fluid (serous or mucous) in the tympanic cavity.

Hydro - ureter. *See* Uterus, Non-Malignant Tumours of (*Pressure Symptoms of Fibroids*).

Hydroxyl.—The radicle OH; water less one atom of hydrogen; it enters into the formation of alcohols, phenols, etc. *See* Physiology, Appendix (*Organic Chemistry*).

Hydroxylamine.—Ammonia in which one atom of hydrogen is displaced by hydroxyl (NH_2OH); it has been used medically as an external application in some skin diseases (*e.g.* lupus).

Hyères. *See* Therapeutics, Health Resorts (*Riviera*).

Hygiama.—A food said to consist of condensed milk, with (fatless) cocoa and cereals added to it; it is recommended for infant and invalid feeding.

Hygiene.—Public Health. *See* Abattoirs; Air, Examination of; Air-borne Diseases; Anthrax; Canal Boats; Cattle; Disinfection; Epidemiology; Food; Hospitals; Infection; Meteorology; Milk; Pregnancy (*Management*); Quarantine; Rabies; Sewage and Drainage; Trades, Dangerous; Ventilation and Warming; Vital Statistics; Water.

Hygroma. *See* Broad Ligament, Diseases of (*Tumours of the Mesosalpinx, Subserous Hygromas*); Bursæ, Injuries and Diseases of (*Bursitis, Acute and Chronic Hygroma*); Neck, Region of (*Cysts, Hygroma of the Neck*); Orbit, Diseases of the (*Cysts, Hygromata*).

Hygrometer. *See* Air, Examination of (*Moisture*); Meteorology (*Humidity, Hygrometry*).

Hygrophila. — The herb known as *Hygrophila spinosa* or *asteracantha* has been used as a diuretic in dropsy and as a urinary sedative; there is a *Decoctum Hygrophilæ* which may be given in doses of ½ to 2 fl. oz.; it is official in the Indian and Colonial Addendum (1900) to the British Pharmacopœia of 1898.

Hymen. *See* Generation, Female Organs of (*External Organs*); Medicine, Forensic (*Rape, Proof of Penetration*); Uterus, Malformations of (*Defects of the Hymen*); Vulva, Diseases of the (*Morbid Conditions of the Hymen*).

Hymenolepsis. *See* Parasites (*Cestodes*).

Hymenoptera. *See* Myiasis (*Insect Stings and Bites*).

Hyoid Bone. *See* Neck, Region of (*Anatomical Details*).

Hyoscine. *See* Hyoscyamus.

Hyoscyamus. *See also* Alkaloids (*Vegetable*); Analgesics and Anodynes; Pharmacology; Toxicology (*Alkaloids, Henbane*). Henbane. The fresh leaves of *Hyoscyamus niger*. They contain two important alkaloids. 1. *Hyoscyamine* occurring as minute crystals, soluble 1 in 120 of water, with an action practically the same as that of atropine. 2. *Hyoscine*, a thick syrupy substance, only used in the form of one of its crystalline salts. It is identical with *Scopolamine*, an alkaloid derived from the root of *Scopolia atropoides*. It has a powerful depressant action on the central nervous system. *Preparations*—1. Extractum Hyoscyami Viride. *Dose*—2-8 gr. 2. Succus Hyoscyami. *Dose*—½-1ʒ. 3. Tinctura Hyoscyami. *Dose*—½-1ʒ. 4. Pilula Colocynthidis et Hyoscyami, containing the green extract of hyoscyamus. *Dose*—4-8 gr. 5. Hyoscinæ Hydrobromidum, identical with Scopolamine hydrobromide; colourless crystals, soluble 1 in 3 of water. *Dose*—$\frac{1}{200}$-$\frac{1}{100}$ gr. 6. Hyoscyaminæ Sulphas, a crystalline deliquescent powder, soluble 2 in 1 of water. *Dose*—$\frac{1}{200}$-$\frac{1}{100}$ gr.

The action of hyoscyamus is very similar to that of belladonna, but it possesses also a sedative effect on the nervous system due to the presence of hyoscine. It is a more powerful peristaltic stimulant than belladonna, and more effectually prevents griping when given in conjunction with

purgatives. For this purpose the green extract is usually employed. It has a marked sedative action on the muscular coat of the bladder, and is given to relieve pain, spasm, and frequency of micturition in cystitis, vesical calculus, prostatitis, etc. It is well to combine it with a urinary antiseptic and buchu or uva ursi. Hyoscine is largely used as a sedative to induce sleep in conditions associated with excitement and restlessness, such as acute mania, delirium tremens, acute alcoholism, and hysteria. It is usually administered hypodermically in doses of about $\frac{1}{100}$ gr., but caution must be observed, as its use is not devoid of danger. It has a certain controlling action on the shaking movements of paralysis agitans, but the condition relapses as soon as it is discontinued. For chorea and epilepsy, and for insomnia due to heart disease, it is not to be recommended. Recently hyoscine has been much used in the treatment of alcohol and drug habits. The patient must be kept thoroughly under its influence, as much as $\frac{1}{100}$ gr. every two hours by hypodermic injection being required in some cases. The statements as to the permanency of the results are somewhat conflicting. It gives very good results in spermatorrhœa and nocturnal emissions in doses of $\frac{1}{100}$ gr. administered at bed-time. Both hyoscine and hyoscyamine have been employed by ophthalmic surgeons for dilating the pupil. They act promptly and in very dilute solution, and the mydriasis does not last so long as that produced by atropine. Recently the use of a combination of scopolamine and morphine has been recommended for general anæsthetic purposes in surgery and midwifery. The employment of large enough doses to produce complete narcosis is to be condemned as very dangerous, but the preliminary injection of $\frac{1}{6}$ gr. of morphia and $\frac{1}{120}$ gr. of scopolamine is not open to this objection, and is said to support the action of ether or chloroform by diminishing nervousness, preventing vomiting, and conducing to prolonged sleep after the operation. In midwifery similar doses are reported to diminish excessive labour pains without interfering with the strength of the uterine contractions. *Duboisine* (*q.v.*), an alkaloid obtained from *Duboisia myoporoides* and usually employed in the form of its sulphate, is chemically and physiologically identical with hyoscyamine.

Hypacusis.—Defective hearing.

Hypæsthesia.—Diminished sensibility or partial anæsthesia of a part.

Hypamaurosis.—Slight or partial amaurosis.

Hypatonia.—Slight atony.

Hyperacanthosis.—Thickening of the prickle-cell layer of the epidermis, as seen in such diseases as tylosis palmæ, akrokeratoma, tyloma, and clavus.

Hyperacidity. *See* Indigestion (*Symptoms, Cardialgia*).

Hyperacusis.—Morbidly increased sensibility to sound. *See* Auditory Nerve and Labyrinth (*General Diagnosis, Tinnitus, etc.*); Tinnitus Aurium.

Hyperadenosis.—Glandular enlargement.

Hyperæmia.—The presence of an increased amount of blood in any organ or tissue of the body; increased vascularity. *See* Ankle-Joint, Region of, Diseases (*Tuberculous Arthritis*); Brain, Affections of Blood-Vessels (*Hyperæmia*); Headache (*Causes, Hyperæmia*); Meninges of the Cerebrum (*Vascular Disturbances, Hyperæmia*).

Hyperæsthesia.—Increased sensitiveness to external impressions of sight, sound, touch, etc. *See* Heart, Affections of Myocardium and Endocardium (*Symptomatology, Palpitation and Cardiac Pain*); Hysteria (*Sensory Disorders, Hyperæsthesia*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms, Nervous, Cutaneous Hyperæsthesia*); Nerves, Multiple Peripheral Neuritis (*Symptoms, Sensory*); Pregnancy, Physiology (*Changes in Nervous System*); Rickets (*Clinical Features*); Tabes Dorsalis (*Symptomatology, Sensory Phenomena*); Visceral Pain (*Cutaneous and Muscular Hyperæsthesia*).

Hyperakusis. *See* Hyperacusis; Tinnitus Aurium.

Hyperalbuminosis.—An excessive amount of albumin in the blood, due to loss of water and salts, as in cholera or after the use of hydragogue cathartics.

Hyperalgesia.—Excessive sensitiveness to painful impressions. *See* Hysteria; Nerves, Multiple Peripheral Neuritis (*Arsenical Poisoning, Symptomatology, Hyperalgesia*).

Hyperaphrodisia.—Excessive venereal desire.

Hyperbulia.—Exaggeration of the power of the will, as it is met with occasionally in insanity; it is used by Friedländer to signify "violent mania occurring in persons of weak will and depraved morals."

Hypercatharsis.—Excessive purging.

Hypercedemonia.—Excessive anxiety.

Hyperchlorhydria.—The presence of more than the normal amount of hydrochloric acid in the stomach. *See* Stomach and Duodenum, Diseases of (*Special Symptomatology, Secretory*).

Hyperchondroma.—A tumour composed of cartilage.

Hyperchromatosis.—Excessive pigmentation, especially in certain forms of skin disease, congenital or acquired (*Auspitz*).

Hypercinesis. — Excessive muscular action; spasm; it is opposed to *hypocinesis.*

Hypercyesis.—Superfœtation.

Hyperdactyly.—Polydactyly. *See* Deformities (*Hand and Fingers*).

Hyperdesmosis.—Increase of connective tissue.

Hyperdicrotism.—When a sphygmographic tracing shows the dicrotic notch lying below the abscissa the pulse is said to show hyperdicrotism. *See* Pulse (*Value of Sphygmogram, Position of Dicrotic Notch*).

Hyperemesis. — Excessive vomiting, such as occurs especially in pregnancy. *See* Pregnancy, Affections and Complications (*Digestive*).

Hyperencephaly.—The teratological type in which there is partial absence of the cranial vault (the occiput is usually present), and the brain lies outside. *See* Teratology.

Hyperepinephry.—A symptom-complex or syndrome consisting of headache, ear symptoms (vertigo, tinnitus), eye symptoms (amaurosis, glaucoma), transitory aphasia and hemiplegia, with arterial hypertension; it is regarded as due to the circulation in the body of too much adrenalin on account of functional superactivity of the adrenal glands.

Hypererethismus.—A state of excessive irritability.

Hyperesophoria. *See* Ocular Muscles (*Abnormal Position*).

Hyperexophoria. *See* Ocular Muscles (*Abnormal Position*).

Hypergensia. — Excessive sensitiveness of taste. *See* Fifth Nerve, Affections of (*Affections of Taste*); Hysteria (*Sensory Disorders*).

Hyperglobulia.—Excess of red cells in the blood; polycythæmia.

Hyperhedonia. — Excessive pleasure experienced in doing agreeable acts (Gr. ἡδονή, pleasure).

Hyperidrosis.—The excessive secretion of sweat. *See* Hysteria (*Disorders of Secretion*); Nails, Affections of (*In Skin Diseases, e.g. Hyperidrosis*); Nerves, Multiple Peripheral Neuritis (*Arsenical Poisoning, Symptomatology*); Skin, Diseases of Sweat and Sebaceous Glands (*Hyperidrosis*).

Hyperinosis. — Excessive amount of fibrin in the blood.

Hyperinvolution. — Superinvolution, *e.g.* of the uterus after delivery. *See* Menstruation and its Disorders (*Amenorrhœa, Atrophy of the Uterus*).

Hyperkeratosis. — Excessive development of the stratum corneum of the epidermis, congenital or acquired. *See* Ichthyosis; Nerves, Multiple Peripheral Neuritis (*Arsenical Poisoning, Symptoms, Hyperkeratosis*); Pregnancy, Intra-Uterine Diseases (*Fœtal Ichthyosis*).

Hyperkinesis. *See* Hypercinesis.

Hyperlactation. — Long-continued suckling, common among the poorer classes, among whom the erroneous idea is prevalent that conception may thus be prevented; it results in a depreciation of the general health and possibly in hyperinvolution of the uterus; immediate weaning is the proper remedy.

Hyperleucocytosis. *See* Immunity.

Hypermetropia. *See* Asthenopia (*Causes, Refractive*); Brain, Tumours of (*Diagnosis*); Glaucoma (*Predisposing Causes*); Refraction (*Hypermetropia*); Retinoscopy (*Hypermetropia*).

Hypermnesia. — Abnormal power of remembering. *See* Memory in Health and Disease (*Hypermnesia or Excitations of Memory*).

Hypernephroma. — A renal tumour, often of great size, growing upon or in the interior of the kidney, derived from "rests" of adrenal tissue.

Hypernœa.—Excessive mental activity (in disease), especially as regards imagination.

Hyperonychia. — Hypertrophy of the nails, *e.g.* in fœtal ichthyosis.

Hyperopia.—Hypermetropia (*q.v.*).

Hyperosmia. — Increased sensitiveness to smell. *See* Nose, Nasal Neuroses (*Hyperosmia*).

Hyperostosis. — Increased growth or hyperplasia of bone. *See* Hypertrophy (*Unilateral*); Rickets (*Clinical Features, Limbs*).

Hyperphalangy. — A structural anomaly consisting in an increase in the number of phalanges in a digit, *e.g.* three phalanges in a thumb and four in one of the other fingers.

Hyperphasia.—The inordinate or excessive talking occurring in some forms of insanity; incoherent garrulity.

Hyperphoria. *See* Asthenopia (*Muscular, Latent Vertical Deviations*); Ocular Muscles, Affections of (*Abnormal Position*).

Hyperphrasia.—"The incoherent and exaggerated forms of speech of a maniacal person" (*Hack Tuke*).

Hyperphrenia.—Extraordinary mental excitement; mania.

Hyperplasia.—Increase in size of any part by increase in the number of its tissue elements. *See* Hypertrophy; Pregnancy, Pathology (*Affections of Ovum and Decidua, Hypertrophy of the Decidua*).

Hyperpneusis.—Excessive flatulence (Gr. ὑπέρ, excessive, and πνεῦσις, panting).

Hyperpnœa.—Deepened respiration, not amounting to dyspnœa. *See* Asphyxia (*Definitions*); Respiration (*Nervous Mechanism, Hyperpnœa*).

Hyperpresbyopia.—Presbyopia of a high degree.

Hyperpyræmia.—That condition of the body, usually met with in middle life, when more nourishment is supplied in the form of food and drink (carbon-containing material) than is needed; it is favourable to the development of gout (*F. Hare*).

Hyperpyrexia.—Extraordinarily high fever (*e.g.* 107° F.). *See* Temperature (*Elevation, Hyperthermia*); *see also* Brain, Tumours of (*Symptoms, Temperature*); Brain, Inflammations (*Acute Encephalitis*); Chorea (*Prognosis*); Hydropathy; Malaria (*Pernicious Attacks, Cerebral Form, Hyperpyrexial Variety*); Meningitis, Tuberculous and Posterior Basic (*Symptoms*); Pneumonia, Clinical (*Rational Treatment*); Rheumatism, Acute (*Treatment of Hyperpyrexia*); Sunstroke (*Definition*); Typhoid Fever (*Symptoms, Temperature*); Typhus Fever (*Period of Advance, Temperature*); Undulant Fever (*Complications, Hyperpyrexia*).

Hyper-resonance.—Excessive resonance on percussion over a part.

Hypersplenomegaly. — Excessive splenic enlargement.

Hyperthermia. *See* Temperature (*Elevation, Causes*).

Hyperthymia.—Mental hyperæsthesia in the form of melancholia (*Hack Tuke*); also insanity evinced by acts of foolhardy daring.

Hyperthyrea.—A diseased state of the thyroid gland, in which its tissues are in a state of active evolution; exophthalmic goitre; it is opposed to the *athyrea* of myxœdema. *See* Thyroid Gland, Medical (*Exophthalmic Goitre*).

Hyperthyroidism.—A state of disturbed action of the thyroid gland, leading probably to hypersecretion; the cause of exophthalmic goitre.

Hypertonia.—Increase in the muscular tone as shown by exaggerated tendon-jerks; muscular irritability. *See* Tendon-Jerks (*Nature*).

Hypertonus.—Excessive tonic contraction of an artery, as shown by thickening of the arterial wall and diminution in the lumen. *See* Arteries, Diseases of (*Arterial Hypertonus*).

Hypertrichosis.—Excessive hairiness. *See* Hirsuties; X-Rays (*Röntgen Ray Therapeutics*).

Hypertrophy.

See also Acromegaly; Fœtus and Ovum, Development (*Decidua*); Gastro-Intestinal Disorders of Infancy (*Congenital Hypertrophy of the Pylorus*); Mammary Gland, Diseases of (*Hypertrophy*); Mouth, Diseases of (*Gums, Hypertrophy of*); Muscles, Diseases of (*Idiopathic Muscular Hypertrophy*); Pregnancy, Physiology (*Local Changes, Hypertrophy of Uterus, Cervix, Abdominal Walls, Breasts, and Heart*); Pregnancy, Pathology of (*Affections of Ovum and Decidua, Hypertrophy of Decidua*); Prostate Gland (*Hypertrophy*); Spleen, Medical Affections of (*Enlargement*); Stomach, Surgical (*Pyloric Obstruction*); Teeth (*Hypertrophy of Pulp*); Thyroid Gland, Medical (*Goitre*).

By hypertrophy is meant an increase in the size of an organ or tissue beyond the ordinary limits, the increase taking place along the lines of physiological growth. It may be defined in the following terms: "Hypertrophy of an organ or tissue expresses that condition where its normal bulk is universally augmented by an additional number or by increased dimension of its tissue elements without deposition of foreign substances" (Hamilton). It may therefore be

called a *progressive nutritive change* (as opposed to a *retrogressive nutritive change*—a degeneration or atrophy), and when it deviates to any great extent from the types of embryonic development or physiological growth we have no longer to deal with hypertrophy, but with a tumour. We can distinguish histologically between true hypertrophy, or increase in the size of the individual tissue elements, and false hypertrophy (hyperplasia), or increase in their number. In the case of hypertrophy of an organ, both true hypertrophy and hyperplasia are commonly associated, but since it is only possible to ascertain the existence of the former by careful micrometric measurements, we generally include both, as stated in the definition given above, under the common term hypertrophy.

Hypertrophy depends on a disturbance of the equilibrium between absorption and consumption of nutriment in favour of the former, a state of matters which is normal during the period of growth—physiological growth being in fact the natural type of the progressive nutritive changes of which hypertrophy is one. Since normal growth depends on three factors: (1) Inherent predisposition on the part of the cells; (2) a due supply of nourishment; and (3) the amount of tissue waste—it is to these we must look in seeking to understand hypertrophy. It is impossible to draw any hard and fast line between simple hypertrophy and overgrowth resulting from inflammation on the one hand, or between it and certain forms of pathological new growth on the other. Further, it is impossible to differentiate sharply between normal development and hypertrophy. Neither does our definition exclude congenital hypertrophy, since there is no reason for discriminating between intra-uterine and extra-uterine growth; it must also include such conditions as simple overgrowth of nails and hair. The influence of the nervous system on the occurrence of hypertrophy is indirect, and would appear to be twofold: (1) it stimulates the cells of the tissues to perform their functions, and, as we shall see, functional activity is in most cases necessary in order that the cells may be capable of assimilating nourishment; (2) as the great controller of the blood-flow it determines the amount of nourishment supplied to the various organs.

Factors influencing Growth and Hypertrophy.—(1) *Inherent Predisposition on the Part of the Cells.*—This varies in the different organs of the body. In some, *e.g.* the thymus, it ceases early; in others—the nervous tissues—it ceases entirely when normal growth is complete; while in others, again—the muscles, glands, and epithelial structures—it persists during the greater part or the whole of life. Speaking generally, however, it is greatest in early life and diminishes with advancing years.

(2) *Supply of Nourishment.*—Increase in the blood-supply alone will not cause overgrowth of all tissues. The conditions under which it may do so are by no means clear. Experimental section of the cervical sympathetic with resulting vaso-dilatation does not by any means invariably cause increased growth in the hyperæmic area. Hunter, however, produced hypertrophy of the spur of a cock by transplanting it into the highly vascular comb. In the human subject we apparently have an example of hypertrophy due exclusively to increased blood-supply in the great development of the corpus luteum of pregnancy as compared with that of menstruation, and occasionally a somewhat similar corpus luteum occurs in the unimpregnated condition, when there is congestion of the pelvic tissues from some pathological cause. In organs whose function is active—the muscles and glands—no amount of hyperæmia alone will cause an increase beyond normal limits. Inflammatory hyperæmia alone, however prolonged, will never cause hypertrophy of either of these structures, but if associated with excessive functional activity it will readily do so. That is, the cells of such organs are only capable of assimilating extra nourishment if they are actively functioning. On the other hand, the supporting structures of the body, and also its epithelial coverings, increase in size as the result of long-continued hyperæmia; their capacity for assimilation, therefore, is mainly if not entirely regulated by the amount of nourishment supplied to them.

(3) *Amount of Waste.*—Diminished waste must be accounted a relatively unimportant cause of overgrowth. Nutrition and waste cannot be regarded as mutually dependent: it does not follow that nourishment will be automatically supplied where waste is excessive, or that where waste is diminished nutrition is also lessened. Each of the processes has its own conditions, and therefore waste must be a factor in determining the size of individual organs. An example of pathological enlargement, associated with diminished tissue waste, is said to be afforded by subinvolution of the uterus, but inflammatory changes also play an important part in its production; excessive development of fat in certain cases of obesity would sometimes fall into this group.

Clinical Varieties of Hypertrophy.—Hypertrophy of an organ may be a purely physiological process, as in the case of the growth of the uterus and mammary glands during gestation. In the pregnant uterus there is both hyperplasia and true hypertrophy, the muscular fibres becoming increased in number as well as in size; according to Kölliker they are eleven times longer and four times broader than in the unimpregnated condition. Excessive development of the voluntary muscles, either as the result of athletic exercises or in consequence of certain occupations, can scarcely be looked

upon as other than physiological; it is, however, essentially the same as any other functional hypertrophy, *e.g.* of the heart in chronic renal disease, the only difference being in the cause which demands the excessive amount of work. As another instance of physiological, or almost physiological, though not compensatory hypertrophy, senile enlargement of the prostate may be mentioned, since, although a cause of disease, it cannot in itself be invariably considered as morbid. If we accept Hamilton's view that the term hypertrophy should be applied to a general but not to a local enlargement of an organ, as for example when we call a local increase of the gland tissue in the breast an adenoma, we may regard cases of enlarged prostate where the middle lobe is specially affected as allied to tumour growth rather than simple hypertrophies.

From a clinical standpoint we may consider hypertrophy under five heads: 1. Compensatory or functional hypertrophy; 2. Hypertrophy due to repeated irritation; 3. Pathological hypertrophy; 4. Unilateral (congenital) hypertrophy; 5. Giantism. Finally, we have to consider what is meant by pseudo-hypertrophy.

1. *Compensatory or functional hypertrophies* occur in many organs as the result of increased work, if there be also an adequate supply of nourishment. The result of excessive functional activity is increased blood-supply, and if the blood contain proper nourishment hypertrophy will ensue; should nourishment be inadequate the organ will become exhausted and cease to function properly, and will probably waste. Compensatory hypertrophy may occur in *paired organs* where one is destroyed or functionally in abeyance for a prolonged period, in *correlated organs* under similar conditions, in *single glandular organs* where a part of the structure is extensively diseased or atrophied, and in *muscular organs* when a mechanical resistance necessitates an increased output of work. Examples of compensatory hypertrophy in paired organs are seen in cases of congenital absence of one kidney, and in cases of removal or long-standing disease of one of the organs. In cases of congenital absence of a kidney the remaining organ may be double its normal size, and when the loss takes place in early life it may attain almost equal dimensions; compensation is less complete after growth has ceased. The enlargement is due to true hypertrophy and hyperplasia of the kidney elements; an increase in the number of the glomeruli, however, occurs only in the case of congenital absence of one organ. An analogous compensatory hypertrophy has been observed in the lungs, suprarenal capsules, testicles, and other paired organs. A functional hypertrophy of the kidneys is sometimes seen in cases of polyuria. Thoma points out that compensatory hypertrophy may occur in paired organs where increased function cannot be assumed by the remaining organ, *e.g.* after excision of one testicle in a newly-born animal. In such cases the hypertrophy may be supposed to depend on nervous influence. It must also be stated that, according to one view, enlargement of a congenitally single kidney is to be looked upon as due to fusion of the two structures. It is also said that the enlargement of the active kidney, when the other is removed or extensively diseased, is due mainly to increase in the lymphatics and dilatation of the blood-vessels. Compensatory hypertrophy of correlated organs is seen in the glandular enlargement which occurs after splenectomy, and also in the enlargement of the pituitary body which has been found after experimental removal of the thyroid. In cases of pressure atrophy of part of the liver (as from tight-lacing) compensatory hypertrophy of the remainder may be observed. Similarly we have enlargement of the part of the thyroid left behind after partial thyroidectomy.

From a clinical point of view the most important group of functional hypertrophies are those found in hollow muscular organs. Any long-continued rise in the blood-pressure will cause thickening of the muscular coats of the arteries and cardiac hypertrophy. The individual fibres of the heart muscle become enlarged, and there is as well a numerical increase. A similar change occurs when the pressure in any chamber of the heart is raised, *e.g.* as the result of valvular disease or obstruction to the pulmonary circulation. Cardiac hypertrophy is, in fact, one of the best examples of the importance of some of the factors we have enumerated. It arises most quickly in young persons, it is seen in greatest perfection in aortic incompetence and Bright's disease, where the work of the heart is enormously increased, and we too often have opportunities of observing its failure when an insufficient quantity of nourishment is supplied, as where the coronary arteries are thickened and partially obliterated.

Another important example of functional hypertrophy is that occurring in the wall of the bowel above the seat of a chronic obstruction of the lumen. This shows itself by the presence of visible peristaltic waves, and by observing the direction in which these pass, and the pattern to which they give rise, we can often form a fairly exact notion of the seat of the obstruction. It is unnecessary to multiply examples of such compensatory hypertrophy; we find it in the stomach in cases of pyloric stenosis, in the bladder in cases of urethral stricture, etc.

Functional hypertrophy of voluntary muscles has already been mentioned. A similar condition may arise from disease. Thus true muscular hypertrophy is met with in Thomsen's disease, where there is increased tonicity of all voluntary muscles; it also occurs in some cases of infantile cerebral paralysis from a like cause. The rare condition described by Gowers as muscular hypertrophy, which is associated with diminished

power and proneness to exhaustion, without alteration in the mechanical or electrical irritability, may also be mentioned. It would appear to be more closely related to the pathological hypertrophies than to the functional type.

2. *Hypertrophy from Irritation.*—Many instances of this will at once suggest themselves. As examples of overgrowth due to recurring pressure or friction, corns, various trade callosities, and milk spots on the pericardium may be cited. Chronic inflammation often results in a hypertrophy of the connective elements in its neighbourhood, as seen in the thickening of the pleura and periosteum after pleurisy and periostitis; the induration of the skin met with in chronic eczema has a similar origin. For the immediate cause of the hypertrophy in such cases we must look to the hyperæmia and consequent increase of nourishment caused by the irritant. We have already seen that while hyperæmia will not cause overgrowth of glandular or muscular organs unless at the same time there is increase of function, that is, unless the cells are stimulated to utilise the increase of nourishment, the cells of the supporting structures of the body, whose function is a passive one, are more directly dependent for their growth on the amount of nutriment supplied to them.

3. *Pathological Hypertrophies.*—In the abovementioned instances the hypertrophy has been, if not physiological, yet in the main beneficial, and its proximate causes, at least, have been fairly recognisable. But we have also to deal with a series of cases in which the hypertrophy is usually harmful, and in which the causes are by no means clear. Thus senile enlargement of the prostate stands on the borderland of the physiological and pathological; it is a common sequel of advancing years, and in some cases it approximates to a tumour, yet, unless it give rise to urinary symptoms, it can scarcely be looked on as a disease. The enlarged thyroid of Graves' disease, the hypertrophy of the pituitary body found in some cases of acromegaly, the enlargement of the spleen found in some cases of anæmia, congenital syphilis, and rickets, the adenopathies of Hodgkin's disease, the hypertrophied thymus found in some infants, which may at this age be the cause of sudden death from suffocation, the hypertrophic changes in the bones accompanying certain chronic lung diseases, and the exostoses of the facial bones in leontiasis ossea, are a few examples of this group. Some of them appear to be primary maladies, others to be the results of the associated disease, while in other cases they are more closely allied to tumour growths.

4. *Congenital hypertrophy*—or, as it is more correctly termed, *hemi-hypertrophy*—is a rather rare condition. In its most typical form it consists of a total hypertrophy of one-half of the body involving the head, trunk, and extremities, the asymmetry being complete in every respect, even extending to the ears, tonsils, two halves of the tongue, and external genital organs. The differences in size may best be illustrated by a few measurements of a case observed by the writer. Ears, 4·5 and 4 cm. long; arms, 18·5 and 17 cm. in length, 15·5 and 13·5 cm. in girth; legs, 25·5 and 22 cm. in length, 24·5 and 17 cm. in girth; mid-sternum to spine at level of nipples, 20·5 and 17·5 cm. The hypertrophy involves mainly the soft parts—the skin, muscles, and connective tissues; the bones themselves, however, are affected in many cases, especially where the length of the limbs is different on the two sides. The overgrowth is as a rule most evident in the more distal parts of the limbs—in the hands and fingers, and feet and toes. The skin is sometimes more active on the hypertrophied side, the hair is thicker and more abundant, the nails are coarser and grow more quickly, and the eruption of the teeth is earlier. The temperature is often, though not always, a degree or two higher on the affected side, and the hypertrophied parts are more susceptible to heat and cold. As a rule the muscular power is greater on the side of the hypertrophy. There are no nervous symptoms, and the internal organs are normal. There remains only one prominent feature of such cases to be mentioned: the presence of nævi and other cutaneous vascular anomalies. These exist in a large proportion of the cases, sometimes in the form of simple capillary nævi, occasionally of larger telangiectases. At times the skin appears normal while the patient is at rest, but when he cries or is excited large patches of cutaneous congestion, differing from nævi only in their temporary duration, are produced. These vascular abnormalities are never strictly unilateral, but are more or less generally scattered over the body. Another not infrequent appearance is a condition of marbling of the skin like that seen in pseudo-hypertrophic paralysis. True congenital hypertrophy is always stationary, the relative size of the parts "being preserved throughout life."

Not all cases, however, conform to the description we have just given. Hemi-hypertrophy is much more commonly limited to one limb, or even to a single digit, than widely spread in its distribution. A large number of cases have been reported where hand and foot alone have been involved. We may regard all these—simple non-progressive hypertrophies, usually strictly unilateral, with preservation of the normal appearance of the parts, without the occurrence of tumours or other deformities, the skin often being the seat of vascular anomalies—as belonging to the group of "true congenital hemi-hypertrophies."

In another group of cases—"false hypertrophies"—a somewhat different condition obtains. In them the abnormality is usually more

limited in extent, but greater in degree. Thus we may have hypertrophy of a hand associated with giant growth and deformity of a finger, with syndactylus, or with tumour growth, usually either lipomatous, or of the nature of an enchondroma. Again, the hypertrophy may lead to ankylosis of the joints, or it may be combined with elephantiasis of the limb. These so-called false hypertrophies also differ from the preceding in not being invariably unilateral, congenital, and stationary; sometimes both feet, or a foot and hand on opposite sides, are affected; the hypertrophy may not develop until childhood or adolescence, it may progress with varying rapidity, with periods of quiescence. Sometimes amputation of a giant finger has been followed by overgrowth of the arm. In fact, false hypertrophies are more closely allied to tumour growth than those just described, and are commonly associated with some other developmental anomaly. Still, the two conditions must not be too sharply differentiated; in true hypertrophy we not infrequently find that one or two fingers or toes have not the same proportionate development as the rest; they may be unduly large or of natural size. In such a case the corresponding digits of the opposite side are sometimes also abnormal.

Greig has proposed an anatomical classification of cases of unilateral hypertrophy. He divides cases into those where the affection is limited to the head and face, and those where it is not so limited. Each group is further subdivided into cases in which the bones only are involved, soft parts only involved, and both bones and soft parts involved. Unilateral hypertrophy limited to the head and face deserves some further notice, and although it is not always congenital may be most fittingly considered here.

Unilateral hyperostosis, or hemihypertrophy limited to the bones of the face, must not be confounded with leontiasis ossea. Greig found that the age of onset varied from three to thirty-five years, the average being fourteen. Four out of fourteen cases collected by him were mentally defective, one was epileptic, and in one there was strabismus. The hyperostosis is sometimes accurately limited to the region supplied by the trigeminal. Hemihypertrophy limited to the soft parts, as well as that in which both soft parts and bones are involved, is usually congenital. In such cases the cheeks and lips are often the places most affected, and the teeth and hair may participate in the overgrowth.

It might be supposed that the distinction between hemihypertrophy and hemiatrophy would not always be easy, but in practice this difficulty does not arise, the hypertrophied side always appearing so abnormally large as to prevent the possibility of error. It is said, however, that hemihypertrophy is sometimes associated with hemiatrophy of the opposite side. Hemihypertrophy must not be confused with the slight degree of asymmetry which is so commonly seen in normal individuals.

The pathological changes in these cases are little known; there seems, however, to be both true hypertrophy and hyperplasia, mainly of the connective-tissue elements. No abnormality of the vascular or nervous systems, except thickening of the arterial walls, has been described. The viscera are normal. The causes of the condition are unknown. Males are more frequently affected than females, and the right side more often than the left. There does not appear to be any hereditary tendency to this, or indeed to any other congenital deformity, in the families of those affected. It seems most likely that it is due to a developmental error in the middle lamina of the blastodermic membrane, but the following theories have also been advanced:—(1) Congenital lesion of the vaso-motor centres, with venous stasis. (2) Partial intra-uterine strangulation of the affected limbs. (3) Congenital defects in the lymphatic system. (4) Fusion of twins. (5) Nervous influences. The distribution of hyperostosis of the face and cranium over the area supplied by the fifth nerve has been alluded to. Greig, considering the comparative frequency with which mental defects are associated with facial hemihypertrophy, especially where the bones alone are involved, believes that the condition is due to an intra-uterine meningitis or cerebritis, which irritates the nerves while their function is yet entirely trophic. He would explain all forms of hemihypertrophy on this hypothesis.

5. *Giantism.*—As is the case in the opposite condition—dwarfism—the nature and causes of giantism are obscure, and its various forms are not well known. Much of the writing on the subject dates from the seventeenth and eighteenth centuries, and, however great its antiquarian interest, is of little scientific value. Probably the largest authentic giants are Frederick the Great's Scottish giant (8 ft. 5 in.), the Irish giant, whose skeleton measures 8 ft. 6 in., and the "Elsässer Bauer," who lacked only half an inch of being nine feet. The often-recurring accounts of men whose stature was ten, twelve, or even fifteen feet must be relegated to the domain of myth.

Of late years, and especially since the recognition of acromegaly as a well-defined disease, which is not infrequently associated with giantism, renewed attention has been directed to the subject. Giantism may be congenital, or it may occur during youth or adult life. Congenital giants are rare, but every now and then children weighing fifteen to twenty pounds come into the world. In such cases the growth during the first year or two is often rapid, and a precocious puberty is reached at five or six years. Thereafter there is premature ageing,

the growth is slowed, and the subjects generally die early.

Giantism may occur in later life under a variety of circumstances, and associated with several other diseases, of which the most common is acromegaly. Sternberg, to whose writings we are largely indebted, divides giants into the following groups:—*A*. Normal giants, without any deformity, who enjoy good health, and reach old age. *B*. Pathological giants. (i.) Associated with acromegaly. (ii.) With multiple tumour-like exostoses, leontiasis ossea, or facial hyperostosis. These often show signs of pressure on the brain or cranial nerves. (iii.) Combined with facial hemihypertrophy (unilateral hyperostosis). (iv.) With multiple curvature of bones, such as scoliosis, bending of the arm bones, genu valgum, etc.—probably osteitis deformans of Paget. (v.) Giantism has also been seen associated with congenital syphilis, tumours of the testicle, and after castration (?).

Since many of the reported cases of acromegaly have occurred in giants, and many museum skeletons of giants, *e.g.* the Irish giant, have been found to show signs of acromegaly, it appears that something more than a merely accidental relationship must subsist between them. Langer, before the recognition of acromegaly, divided cases of giantism into those with normal skulls, and those who had an enlargement of the hypophysis. The latter he called pathological, and many of them are now known to have been the subjects of acromegaly. It has been said that all giants are acromegalians, but the incorrectness of such a statement is obvious from the above grouping of the cases. Another view of the relation between the two conditions is this: that when the disease occurs in youth, giantism results; when in later life, acromegaly. We now know, however, that typical acromegaly may develop in young persons. It appears that while about 40 per cent of all giants suffer from acromegaly, only about 20 per cent of all acromegalians are giants—that is, acromegaly is more common in giants than *vice versa*. As Sternberg's classification shows, we may have along with giantism not only acromegaly, but a variety of other more or less rare diseases of the bones—leontiasis ossea, osteitis deformans, etc., and it has been argued that giant growth may predispose to the occurrence of such widely-spread dystrophies and lesions of the supporting structures of the body. Much further observation is, however, required before the relations of giantism to acromegaly and these other diseases can be known.

Pseudo-Hypertrophy.—In pseudo-hypertrophy of muscles there is not merely a replacement of atrophied muscle-fibres by fat, but there is actual hypertrophy of the fat and connective tissue. The affected muscle-fibres are abnormally small; most of them, however, preserve their normal striation, but where the change has progressed farthest they show granular degeneration and other changes. The condition is a primary disease of the muscles due to an inherited tendency.

Treatment.—Little need be said on this point. Since compensatory hypertrophy is in the main beneficial, and is, in fact, what we aim at in dealing with the conditions to which it is due, we have to do our best to further it in most cases. The indications are furnished by our knowledge of its proximate causes. We endeavour to supply appropriate nourishment, to adapt the stimulus to the capability of the hypertrophying structure—as for example when we order rest in a case of cardiac valvular disease in order to give the ventricular walls time to gain strength and cope with the effects of the lesion, and finally (though this is less often within our power) to diminish tissue waste. Apart from this, compensatory hypertrophy, as such, requires little or no treatment. In the case of the morbid conditions grouped under the heads of pathological and inflammatory hypertrophies, the treatment—further than if possible removing the cause—will vary according to the organ affected. Reference must be made to the special articles for particulars. Hemihypertrophy does not admit of much treatment. Benefit is said to have followed the application to the limbs of elastic pressure so as to retard growth. In some cases surgical measures may be needed.

Hyperymenoma.—A tumour consisting of membrane (Gr. ὑπέρ, excessive, and ὑμήν, a membrane).

Hyphæ. *See* Micro-Organisms (*Hyphomycetes or Moulds*).

Hyphæma or **Hyphæmia.**—Hæmorrhage into the interior of the eye. *See* Eyeball, Injuries of (*Detachment of Iris*); Iris and Ciliary Bodies (*Inflammatory Conditions*).

Hyphedonia.—Morbid lessening of the feeling of pleasure in doing agreeable acts; it is opposed to hyperhedonia (*q.v.*).

Hyphomycetes. *See* Micro-Organisms (*Hyphomycetes*).

Hypinosis.—Deficiency of fibrin in the blood, as opposed to *hyperinosis* (*q.v.*).

Hypnacetin.—An antiseptic and hypnotic drug; it is an acetophenon and phenol derivative, and it has been given in doses of 3 or 4 grains.

Hypnagogic State.—That condition in which an individual is neither awake nor fully asleep. *See* Sleep, Normal and Morbid (*Hypnagogic State*); Unconsciousness (*Double Consciousness*).

Hypnal.—A preparation of chloral hydrate and antipyrine, said to act as a hypnotic and analgesic (*Bardet*).

Hypnalgia.—Pain coming on or recurring during sleep (Gr. ὕπνος, sleep, and ἄλγος, pain).

Hypnenergia.—Somnambulism (*q.v.*).

Hypnobadisis.—Somnambulism (Gr. ὕπνος, sleep, and βάδισις, walking). *See* Hypnotism; Sleep, Normal and Morbid; Unconsciousness.

Hypnogen.—A hypnotic medicine, being a purified form of diethyl-barbituric acid; it is sparingly soluble in water, and is given in doses of 5 to 15 grains in the form of palatinoids (*Oppenheimer*).

Hypnosis. *See* Hypnotism.

Hypnotics. *See also* Chloral; Paraldehyde; Pharmacology; Sulphonal; Trional, etc. A true hypnotic is a remedy which, when given in quantity sufficient to induce sleep, does so without disturbing the relationship of the mental functions to the external world; a narcotic not only induces sleep, but profoundly disturbs this important relationship. The *rôle* of these remedies should be carefully differentiated, and it is well to recognise the fact that the injudicious use of the former exerts a distinct influence towards the indulgence of the latter more dangerous remedy. If this were more thoroughly appreciated, we would meet with fewer cases of morphinomania, chloral and cocaine habit than unfortunately pertain at present. For the induction of sleep the following points may be necessary:—

I. To produce a slight degree of anæmia of the cerebrum, *e.g.* by dilating blood-vessels in other parts of the body, or by stilling any excited cardiac action.

II. To lessen the functional activity of the organism as a whole, and especially in certain cases, *e.g.* that induced by derangement of the digestive tract (as by a too heavy late supper, or a dinner indulged in not wisely but too well), and also that induced by pain or discomfort in some peripheral area. A consideration of these points shows us that a hypnotic may act in one of two ways:—

1. By an indirect action in removing the cause that is preventing sleep.

2. By a direct action—that of a paralysing action on the cerebrum.

Most of the hypnotics in common use belong to the latter class; some of these, *e.g.* the bromides, have a slight action both as direct and indirect hypnotics. The use of hypnotic substances is greatly abused, and for this state of affairs medical men are in part responsible. Not by any means infrequently do we meet with patients complaining of more or less minor ailments, whose history reveals them to have been the slaves of sulphonal or chloral hydrate, taken indiscriminately for many months or even longer, and the treatment of such cases is much more difficult in virtue of that habit. It is a wise rule to observe that in a considerable proportion of those cases in which the administration of a hypnotic is really called for, one should be chosen which there is little prospect of the patient taking a particular fancy to. Herein paraldehyde is valuable. But we must bear in mind that with no class of medicinal remedy is there a greater tendency to idiosyncrasies on the part of patients, and these idiosyncrasies are usually of a nature calling for special vigilance on the part of the practitioner. It may be that the dose given was insufficient, and in place of a paralysing action in the cerebrum, the very reverse action was induced, to the detriment both of the patient and practitioner. Again, the dose may have been excessive, and a resulting depression of the heart and vital powers causes additional anxiety.

Special care must be taken with the use of all recent remedies with advertised powerful hypnotic action, said to be unaccompanied by any such untoward effects. A few general rules can only be given; no hard and fast rules can be formulated.

1. Ascertain the cause of the sleeplessness, and if, as is very frequently the case, that depends on a general state of health, calling for a short or long complete rest with change of air, scenery, occupation, etc., that must be enjoined, and to the exclusion of all so-called hypnotic remedies.

2. General tonic remedies, and it may be special tonics, such as digitalis, are very frequently the line of treatment indicated.

3. In cases of brain unrest and irritability resulting from brain fatigue, in addition to enjoining the necessary brain rest, a judicious course of bromides will be found most appropriate. The following prescription can be cordially recommended:—

℞ Sodii bromidi . .	℥ss.
Quin. hydrobrom. . .	ʒss.
Tinct. aurantii . .	℥j.
Aquæ ad . . .	℥vj.

A tablespoonful to be taken in a wineglassful of water an hour before bed-time. For use under medical directions only.

4. In other cases it may be advisable to fall back on sulphonal, bromidin, chloretone, trional, and the like—in the use of which two points should always be kept in mind:—

(*a*) The remedy should be prescribed for a stated time, and only taken under medical supervision.

(*b*) The dose administered should be carefully selected to meet the requirements of each case; the great variations in susceptibility of different patients must ever be kept in mind.

Hypnotism.

See also Alcoholism (*Treatment, Breaking the Habit*); Catalepsy (*Differential Diagnosis*); Colour Vision (*Disturbance of Colour Perception*); Dipsomania (*Treatment*); Hysteria (*Treatment, General*); Malingering (*Curative Means*); Memory in Health and Disease (*Periodic Loss of Memory, Diagnosis*); Stammering (*Treatment*).

Introduction.—Hypnotism, although officially recognised, can hardly be described as well known. Hence facts must be cited and explanations given which would be out of place in dealing with more familiar departments of medicine. For those who wish to understand, and possibly to practise, hypnotism, something more is required than a mere account of cases which have been relieved or cured by it. Information on many points is necessary, as the questions put to me show, and to these the following article is an attempted reply.

Historical

Just as chemistry arose from alchemy, astronomy from astrology, so hypnotism had its origin in mesmerism. Hence, among hypnotic phenomena are to be found those mesmeric ones which have stood the test of rigorous investigation, and are now explained in a more scientific way. Phenomena such as Mesmer (*b.* 1734, *d.* 1815) described had been observed from an early date in human history, but after his day they were usually called by his name, and explained by his theory, *i.e.* by the action of a mysterious force or fluid which emanated from the operator.

In 1837 a great mesmeric revival began in England, of which John Elliotson was the prime mover. After seeing the experiments of Dupotet, Elliotson introduced mesmerism into his hospital practice and teaching. Shortly afterwards, however, this was forbidden by the hospital authorities, whereupon he resigned his appointments and continued his work independently. Mesmeric infirmaries were opened in London, Edinburgh, Dublin, and elsewhere, thousands of patients treated, and hundreds of painless operations, including amputations of the thigh, etc., performed.

Elliotson's influence spread to India. There James Esdaile, surgeon to the East India Company, heard of his work, and, in 1845, tried to mesmerise a Hindoo convict about to undergo a surgical operation. The result was successful, and, at the end of a year, Esdaile reported 102 painless operations to the Government. Shortly afterwards he was appointed to a Government hospital in Calcutta, so that he might further test mesmerism as an anæsthetic. Until he left India, in 1851, Esdaile occupied similar posts, and recorded nearly 300 painless major operations and many thousand minor ones.

The mesmeric work of Elliotson and Esdaile was bitterly opposed by the medical profession, and the movement practically ceased at Elliotson's death.

James Braid (born 1796, died 1860) commenced his mesmeric investigations in November 1841, after seeing Lafontaine produce apparently genuine phenomena. Braid demonstrated the subjective origin of mesmeric phenomena, substituted the term hypnotism for that of mesmerism, invented the terminology which we still use, and employed hypnotism with good therapeutic results. He published many works on the subject, all now out of print, and most of them little known. A list of those which I have been able to trace, thirty-eight in all, is to be found in the *Revue de l'Hypnotisme* for June 1898.

In 1859, Azam of Bordeaux became acquainted with Braid's work, and commenced to investigate the subject. About the same time, Broca and Velpeau drew the attention of the Académie des Sciences to Braid's researches. The interest thus excited soon faded, and it was not until 1883 that *Neurypnology*—Braid's first published book—was translated by Jules Simon. Despite the fact that Braid's theories were adopted by such well-known men of science as Professors Carpenter and John Hughes Bennett, the practice of hypnotism apparently ceased in England at his death.

In 1860, Liébeault began to investigate mesmerism, and, four years later, started his gratuitous practice of hypnotism amongst the poor of Nancy. He published *Du sommeil et des états analogues considérés surtout au point de vue de l'action de la morale sur le physique* in 1866, but of this one copy alone was sold, and Liébeault's work was ignored by the profession until Bernheim became a convert in 1882, and employed hypnotism in his hospital practice. In 1884, Bernheim published *De la suggestion dans l'état hypnotique et dans l'état de veille*, and, in 1886, *La thérapeutique suggestive.* Liébeault's name now became well known, and doctors flocked from all countries to study his methods.

Independently of Liébeault, Charles Richet, in 1875, drew attention to the genuine nature of hypnosis, and termed the condition *somnambulisme provoqué.* In 1878, Charcot began to lecture on the hypnotic phenomena observed in

hystero-epileptics. In Germany, Heidenhain and others commenced to investigate the subject in 1880, but the movement they originated soon ceased. Charcot and his followers formed the school of the Salpêtrière; their views, however, are now generally discredited, and have done little or nothing to further the therapeutic use of hypnotism. On the other hand, the movement originated by Liébeault has now spread over Europe, its development in Germany being mainly due to Forel. In England, although Braid was never completely forgotten, the hypnotic revival, apart from my own share in it, arose from Liébeault's influence, and hypnotism, despite some opposition, has now gained a recognised place.

At the Birmingham meeting of the British Medical Association in 1890, a committee appointed to investigate hypnotism reported unanimously, two years later, that they had satisfied themselves as to the genuine nature of hypnotic phenomena, and the value of hypnotism as a therapeutic agent. Hypnotism has now definitely taken its place in medicine and science. No work on general therapeutics is complete without reference to it, and the physiologist or psychologist who ignores its existence, or denies its phenomena, imperils his reputation.

Methods of Inducing and Terminating the Hypnotic State

Inducing Hypnosis.—The chief methods of inducing hypnosis are—(1) Sensory stimulation, *e.g.* passes with contact, fixed gazing, etc.; (2) Central stimulation by suggested ideas. The first was the only one consciously employed by the mesmerists; the latter is the avowed method of the Nancy school. The modern hypnotiser, however, borrows in technique from Mesmer and Liébeault with equal impartiality. The members of the Nancy school, while asserting that everything is due to suggestion, do not hesitate to use physical means, and Mesmer's passes are almost exactly reproduced by Wetterstrand. Fixed gazing generally precedes or accompanies suggestion, and, when these fail, Bernheim has recourse to narcotics. At first Braid relied mainly on fixed gazing; later, he hypnotised as readily in the dark as in the light, and easily succeeded with the blind. As the result of this, he concluded that the influence acted directly on the mind—not through the optic nerves—and that the best method was *direct verbal suggestion.* While it is doubtful whether physical methods alone have ever succeeded, hypnosis can certainly be induced when these methods are excluded. Here, the subject is made to understand the condition the operator wishes to induce, and to expect its appearance. This condition is kept constantly before the subject's mind by the operator's reiterated verbal description of it; nothing more is necessary. Once hypnosis has appeared, it can be reinduced at any time in response to any signal previously arranged by suggestion.

Terminating Hypnosis.—No matter how profound the hypnosis, the subject can always be aroused with ease. It is only necessary to suggest during hypnosis that the condition shall terminate in response to some given signal, such as the audible counting of "one," "two," "three" by the operator. Afterwards the subject invariably responds at once whenever the signal is given.

Experimental Phenomena of Hypnosis

These vary with the depth of the hypnosis, the personality of the subject, and the methods of the operator. In the earlier stages we do not find all the phenomena which characterise the deeper ones, but in the latter we can induce not only its own phenomena, but those of all lighter stages. For convenience' sake, therefore, I propose to describe the phenomena which have been evoked in the most profound form of hypnosis, *i.e.* somnambulism.

Changes in the Voluntary Muscles.—The following muscular phenomena have been observed:—*Catalepsy.*—In extreme cases there is general tonic contraction of the muscles. *Flexibilitas Cerea.*—Here the limbs can be bent like soft wax and maintain any position in which they are placed. *Increase of Muscular Power.*—Sometimes the subjects are able to perform physical feats far beyond their normal strength. *Paralysis.*—The different voluntary muscles may be paralysed singly or in groups. In some cases, the muscles necessary for the performance of a given movement do not contract; in others, the muscles act, but are overpowered by the violent contraction of the antagonistic ones. There are, however, few, if any, changes in the voluntary muscles which are absolutely characteristic of hypnosis, and none which arise independently of direct or indirect suggestion. The most noticeable phenomenon is the long maintenance of an uncomfortable posture, associated with extreme muscular rigidity, without subsequent fatigue.

Changes in the Involuntary Muscular and Vaso-motor Systems.—*Pulse.*—In many instances the frequency of the pulse can be easily altered by suggestion, this being accompanied by corresponding sphygmographic changes. Thus, increased speed is associated with decreased tension, and *vice versa.*

Bourru and Burot, Mabille, Jules Voisin, Artigalas, Rémond, Hulst, etc., report cases of *bleeding* from the skin; Forel, Schrenck-Notzing, Rybalkin, etc., *local redness of the skin;* and Focachon, Rybalkin, Krafft-Ebing, Hulst, etc., *blisters* produced by suggestion. Krafft-Ebing and others cite cases of *alterations of temperature.*

The evidence as to *local redness of the skin* and *bleeding* is convincing in some instances and

unsatisfactory in others. As to the alleged *changes of temperature*, further and more careful observations are necessary, while the evidence as to *blistering* is not conclusive.

Forel and other observers have arrested and excited *menstruation* during hypnosis; caused its appearance at a given date by post-hypnotic suggestion, and regulated its duration and intensity. The action of the *bowels* can be easily excited by suggestion. In short, many cases are cited which tend to show that *secretion* and *excretion* are largely under the influence of suggestion.

Changes in the Special Senses, Common Sensation, Muscular Sense and Appetite.—*Vision*, both distant and near, can be altered by suggestion. In some instances, at all events, this is due to changes in the muscles of accommodation. Many instances of suggested hyperæsthesia of the sense of *smell* are recorded. An increase of *muscular sense* is shown by greater power of discrimination between minute differences in weight, and in other ways. *Common cutaneous sensibility* can be increased, the subjects sometimes distinguishing the points of the compass at half the normal distance. *Thermo-sensibility* can also be rendered more acute. *Hunger* and *thirst* can be excited, or temporarily arrested, by suggestion.

The activity of the *special senses* can be decreased or arrested. A psychical *dumbness*, *blindness*, or *deafness* can be produced, while, in deep hypnosis, *analgesia* and *anæsthesia* can be easily evoked, either simultaneously or separately. Cases in which impressions, which otherwise would have aroused consciousness, are arrested before they reach the higher cerebral centres are usually classed as *negative hallucinations*. The term *positive hallucination* is applied to cases where the memory of past sensory impressions has been aroused by suggestion, and the subject led to believe in their reality.

Automatic Writing.—Problems, suggested to the subject during hypnosis, may be recorded after waking by automatic writing. Here, the primary waking consciousness retains no recollection of the hypnotic suggestion. It does not know that the secondary consciousness, after the hypnotic state has been terminated, first solves the problem and then directs the motor acts which record it. It is also unconscious of the motor acts themselves.

Hypnotic and Post-Hypnotic Appreciation of Time. — Many observers have noticed an increased appreciation of time during hypnosis and in the fulfilment of post-hypnotic suggestions. One of my subjects, if told to do something at the expiration of a complicated number of minutes from a given hour, as, for example, 20,845, almost invariably carried out the suggestion with absolute precision, while no recollection of the command or its execution existed in the primary consciousness. Further, the calculations involved were far beyond the subject's normal arithmetical powers, while her waking self possessed no special power of time appreciation.

Rapport.—The mesmerists of Elliotson's day believed that a peculiar *rapport* existed between the operator and his subjects. The latter were supposed to be in relation by their senses with those who mesmerised them, but with them alone. Braid pointed out that the condition was a purely artificial one, the result of training, and that, even when it existed, the *rapport* was more apparent than real, as outsiders were able to get into relationship with the subject, although they had to employ special devices for this purpose. Moll's later observations confirm Braid's views.

Memory.—In slight hypnosis there is absolutely no alteration in memory, but in the most profound state—somnambulism—there is post-hypnotic amnesia, *i.e.* the subject during normal life remembers nothing of what had taken place in hypnosis. On being rehypnotised, however, he can recall all the events of previous hypnoses, those of normal life he had previously been able to remember, and frequently also many things which the waking self had forgotten.

Volition. — I have never, either in this country or abroad, seen a hypnotic suggestion carried out which involved anything opposed to the patient's prejudices, feelings, or moral sense. Bernheim and others base the possibility of hypnotic crime on the fact that hypnotic subjects will sometimes execute imaginary ones, *e.g.* put a lump of sugar into a friend's teacup, when told this is arsenic. Questioning in subsequent hypnosis, however, always reveals the fact that the subject knew exactly what he was doing, and fully realised the experimental nature of the transaction.

Self-Hypnosis.—Once genuine hypnosis has been induced, it is an easy matter to teach the subject to evoke the condition for himself. Afterwards, he can make the necessary suggestions to himself while awake, and reproduce the state at a given signal. Some of my former patients still employ hypnotism in this way for the relief of pain, the production of sleep, etc.

The Therapeutic Uses of Hypnotism

With the space at my disposal, it is only possible to give a short outline of the therapeutic uses of hypnotism, while illustrative cases must necessarily be few in number and lacking in detail. It is in functional nervous disorders that hypnotism yields its best results. Thus, Bérillon and many others assert that it is specially indicated in the following hysterical affections:—

I. *The convulsive attacks of "grande hystérie" and the symptoms which sometimes persist after them—paralysis, contracture, anæsthesia, etc*

In this country I have only seen three cases of "grande hystérie" of the true Salpêtrière type. The first, treated by drugs alone, died from cerebral hæmorrhage following a convulsive attack. The second, hypnotised after repeated failures, recovered completely and has remained ten years without relapse. The third, after varied treatment, passed four months in the National Hospital, Queen's Square, where subcutaneous injections of hyoscine, and large and frequently repeated doses of chloral, bromide, etc., were administered, her condition growing distinctly worse meanwhile. On 14th June 1900, when I first saw her, she was unable to walk; there was a contracture of the muscles of the left leg, constant clonic muscular spasm of the muscles of the head, trunk, and extremities on the left side, together with frequent and violent generalised convulsive attacks of the true Salpêtrière type. On June 18th, she was discharged from the hospital and came under my care; she commenced to improve almost immediately under hypnotic treatment, and three weeks later all morbid symptoms had disappeared.

Numerous cases of "grande hystérie," with good results, are reported by Continental observers, and Bérillon, who for three years watched Dumontpallier's cases at the Pitié Hospital, states that most of the patients made a complete recovery and are now in good health —some married and mothers of families, others occupying responsible business positions.

Voisin reported a case of long-standing hystero-epilepsy with maniacal attacks; the patient recovered and became wardrobe maid at the Salpêtrière.

II. *Monosymptomatic hysteria — monoplegia, mutism, aphonia, hiccough, cough, blepharospasm, discromatopsy, rhythmical chorea, etc.*

The following are illustrative cases from my own practice:—

Mrs. ——, aged 49; singultus of three years' duration, supposed to be due to large uterine fibroid. No improvement from careful drugging, electrical treatment, etc. Hypnotised. Recovered. No relapse after five years.

Miss ——, aged 20; aphonia of four years' duration, with loss of reflex sounds. Paresis of lower extremities. Rapid recovery under hypnotic treatment.

Miss ——, aged 19; violent muscular tremor of extremities of fifteen months' duration. Careful hospital treatment without benefit. Hypnotised. Rapid and complete recovery. No relapse after five years.

Bérillon, de Jong, Wetterstrand, van Eeden, van Renterghem, and many other Continental observers, report successful cases of hysterical aphonia, hiccough, cough, mutism, blepharospasm, chorea, etc.

Dr. A. Gros d'Apt reports a case of hysterical paralysis of the lower extremities of three years' duration, which was rapidly cured by suggestion.

Van Renterghem records a case of long-standing clonic spasm of the cervical muscles in a man, aged 42, who had been treated without success by drugs, electricity, hydropathy, nerve-stretching, etc., but completely recovered under hypnotic treatment.

A similar successful case is reported by Tatzel. The patient, a man, aged 44, had been compelled to abandon work and, after having spent all his money on medical and surgical treatment, ended in the workhouse. Tatzel also reports two successful cases of paramyoclonus multiplex, while Lemoine cites one of hysterical tremor of twenty-one years' duration, simulating paralysis agitans, which recovered after six days' hypnotic treatment.

III. *The various manifestations of ordinary hysteria — insomnia, dyspepsia, visceral and menstrual troubles, neuralgia, etc.*

The following are illustrative cases from my own practice:—

Miss ——, aged 20; insomnia from infancy, with spinal neuralgia of ten years' duration. Complete recovery after short hypnotic treatment. No relapse after five years.

Mrs. ——, aged 41; migraine, dysmenorrhœa, dyspareunia, chronic constipation, dyspepsia, insomnia, constant depression, spinal neuralgia, etc. Slight hypermetropia, over-corrected by ciliary spasm, causing virtual myopia—could only read third line of Snellen's unaided. All the morbid symptoms, including the ciliary spasm, disappeared after a short hypnotic treatment, and the patient was able to read the bottom line of Snellen's unaided. The ophthalmic observations were made by the late Mr. Bendelack Hewetson. Two years later there had been neither relapse nor return of the ciliary spasm.

Miss ——, aged 28; pain in hip and leg of five years' duration, with inability to walk. Treatment: drugging, rest on back for two months; Weir-Mitchell; massage; electricity; hydropathy; Paquelin's cautery to leg—about 20,000 applications in all. Meanwhile, the patient grew steadily worse; all treatment was then abandoned, and she was considered incurable. Complete recovery followed two days' hypnotic treatment; the patient exchanged her bath chair for a bicycle, and now, four years' later, is well, active, and strong.

As a large proportion of my patients suffered from hysteria, I could cite many cases similar to those just given. In all the type was identical, the details alone differed. One or more of the bodily functions was performed consciously and painfully, and some form of headache or neuralgia was generally present, together with more or less marked symptoms of mental instability.

On the Continent, hysteria has also been the disease most frequently treated by hypnotism,

and successful cases of every known form have been reported. These are so numerous that it is impossible here to give even a *précis* of them. (See also "Hysteria, Treatment of," p. 321.)

IV. *Mental troubles of a hysterical nature. Perversion of sentiment, obsessions, irresistible impulses, hallucinations, melancholia, etc.*

Perversions of sentiment are of common occurrence in hysterical patients, and are generally associated with insomnia, depression, uncontrollable weeping, etc. The changes in sentiment vary from indifference or dislike for those formerly loved, up to a more or less intense hatred, sometimes associated with impulses to injure the person disliked. Once hypnosis is induced, such cases generally quickly recover.

Hypnotic treatment often gives good results in cases of obsession. The following are some amongst the varieties which have come under my notice:—Involuntary contraction of the bladder, arising solely from a fixed idea as to urination, and independently of distension and other physical causes; dread of cancer, followed by delusion as to its presence; fear of insanity, associated with insomnia and melancholia; excessive blushing, with fixed idea of being constantly noticed and adversely criticised; fixed idea of being haunted, with delusions as to visits from the spirit of a deceased enemy; fixed idea as to the possibilities of having injured others accidentally, with constant *folie du doute* and *délire du toucher*, together with suicidal impulses and more than one attempt at suicide; fixed ideas as to business incompetence, with resultant withdrawal from society; morbid fears as to death, accident, loss of employment, associated with agoraphobia, delusions, hallucinations, and suicidal impulses; dread of thunderstorms; claustrophobia; dread of travelling by rail or carriage; fixed idea that something dreadful would happen to the Almighty if anything were done by the patient contrary to various forms of popular superstition; fear of catching syphilis, with *folie du doute* and *délire du toucher* associated with constant washing of the hands and cleaning, or destruction, of personal effects; impulse to touch certain objects, with dread of some fearful catastrophe if this were omitted; fixed idea of sexual incapacity, associated with consequent impotence. All the cases just referred to recovered, while some of them have passed ten years without relapse.

The mental condition in the above cases varied widely. In many instances the patients fully recognised that the fears or ideas which tormented them were absurd and groundless. In others the morbid ideas were more or less completely assimilated, the fears had become delusions, and were often associated with hallucinations.

Successful cases of obsession are also reported by Schrenck-Notzing, Hecker, Wetterstrand, van Eeden, de Jong, van Renterghem, Bernheim, Delbœuf, Gorodichze, Russell Sturgis, Voisin, Burot, Mavrokakis, Bourdon, etc.

Vicious and Degenerate Children.—At the International Congress of Hypnotism, Paris, 1889, the therapeutic value of suggestion in the following diseases of children was generally recognised: Incontinence of urine and fæces, nervous "tics," nocturnal terrors, chorea, onanism, blepharospasm, and many other disturbances of the nervous system of a functional character. Further, that suggestion was an excellent auxiliary in the education of vicious and degenerate children, especially where there were habits of lying, cruelty, theft, inveterate idleness, cowardice, or imbecility.

I have seen several children—who undoubtedly suffered from moral insanity—recover under hypnotic treatment, and some of them (after the lapse of many years) show no symptoms of the original disease. Some were dirty, insolent, violent in temper, and addicted to self-abuse and purposeless theft, and, in addition, suffered from nocturnal terrors, insomnia, and various "tics."

According to Bérillon, hypnotism is not only useful in moral perversity, but also forms the best method of treating various nervous tricks. He draws special attention to the frequency of nail-biting in children, and to the fact that it is often associated with other marks of degeneracy. Such conditions can be successfully treated by suggestion; and I have seen instances of nail-biting, even when the habit has been long established, that have disappeared after a single hypnotic *séance*.

Liébeault records 77 cases of enuresis nocturnalis, 45 being boys and 32 girls. The youngest was three, the oldest eighteen years of age, the average being about seven. In all, with the exception of 9, the malady dated from birth. Of these, 56 recovered, 9 were improved, while in 12 there was no alteration. In my own practice, the results, though not so striking as those of Liébeault, have been eminently satisfactory.

Neurasthenia.—Schrenck-Notzing has published a more or less detailed account of 228 cases of neurasthenia treated by hypnotism, and the following, amongst others, report successful cases, Brügelmann, Bérillon, Bourdon, Voisin, Burckhardt, Forel, Ringier, Ritzmann, Bourru, Burot, Stadelmann, von Corval, Michael, Drozdoivski, von Kozuchowski, Neilson, Bernheim, van Renterghem, Wetterstrand.

According to Schrenck-Notzing, the sexual perversions so often associated with neurasthenia, even when these have changed the entire personality, are frequently cured by suggestion.

Insanity.—According to the late Dr. Auguste Voisin, of the Salpêtrière, no one believed it was possible to hypnotise the insane until 1880, when he first succeeded in a case of acute mania. From

that date, until his death, Voisin devoted himself largely to the treatment of mental disorders by hypnotic suggestion; and, while he admitted that hypnotism could do nothing in somatic mental affections, such as general paralysis, cerebral softening, etc., stated that he had cured many cases of undoubted insanity, as well as other disorders more or less closely allied to it, *e.g.* melancholia with hallucinations, suicidal impulses, and delusions of persecution; acute and subacute mania; hysteria and epilepsy; morphinomania; dipsomania; obsessions, such as *délire du toucher*, agoraphobia, claustrophobia, etc. Voisin regarded hypnotic suggestion as extremely valuable in mental disorders, and often obtained good results with comparative facility once hypnosis had been induced, a fact which caused him to express his regret that all the insane were not hypnotisable.

Dr. Répoud (Cantonnal Asylum of Marsens, Fribourg) claims to have cured cases of undoubted insanity, while successful cases are also reported by Burckhardt (Préfargier Asylum), Liébeault, Brémaud (Brest), Velander (Yönköping, Sweden).

In 1890, Drs. Percy Smith and A. T. Myers published an account of the hypnotic treatment of twenty-one insane patients in Bethlem Hospital. Improvement followed in six instances, but the results appear to have been due more to increased personal attention than to hypnotic influence.

Dr. George Robertson, in 1893, hypnotised and controlled the worst case of suicidal and homicidal mania there had been in Morningside for ten years. He concluded that, in insanity and allied disorders, hypnotism was of service in producing sleep, quietening the brain, and preventing outbursts of excitement from passing into mania; also in dispelling fleeting delusional states and minor psychoses, as well as for purposes of management.

In 1897, Dr. Woods, Hoxton House Asylum, reported good results in fifteen cases of insanity; the majority of these, however, apparently belonged to minor forms of melancholia.

Personal Results.—With very few exceptions my results in cases of *undoubted* insanity have been disappointing. In many instances I have failed to induce hypnosis, while in others, although I was able to produce sleep and relieve the pain of organic maladies, the suggestions were entirely without effect upon the insane ideas. There were, however, some favourable exceptions, *e.g.* a case of acute mania with attempted suicide, followed by mutism, insomnia, violence, and persistent refusal of food. The patient, who had been certified, was in an asylum, and all feeding was forcible. After the first *séance* forced feeding was never necessary, and the patient made a complete recovery.

I have also obtained good results in subacute puerperal mania with suicidal impulses, and in cases of melancholia of a more or less hysterical type. In some of these cases the patients had previously been certified.

Voisin's claim to have been the first to employ hypnotism in the treatment of the insane is untenable. In 1852, Esdaile hypnotised thirty-seven insane patients at the Calcutta Asylum, but admitted that the therapeutic results were disappointing. About the same date, Dr. Keen (Berhampore Asylum, India) found hypnotism of much service in procuring quiet and maintaining discipline, thus forestalling the observations of Dr. Robertson. Braid and Elliotson also reported cases of insanity cured by hypnotism.

Dipsomania.—Hypnotism yields excellent results in dipsomania. Amongst my successful cases, of both sexes, the duration of the disease had varied from five to seventeen years; some had had several attacks of delirium tremens and epilepsy, and in most there was a family history of alcoholism. In many instances repeated and prolonged treatment in retreats had failed, while the "gold cure" had been equally unsuccessful. Some of the earlier cases have now remained over ten years without relapse.

Forel claims to have obtained excellent and durable results in dipsomania, and successful cases are also reported by Voisin, Ladame, Tatzel, Hirt, Neilson, de Jong, Liébeault, Bernheim, van Eeden, van Renterghem, Wetterstrand, Schrenck-Notzing, Krafft-Ebing, Hamilton Osgood, Cruise, Tuckey, Woods, etc.

Much depends, however, upon the patient's mental attitude and the operator's management of the case. The following are the more important points:—

1. The patient must be willing to be cured. Some, amongst my earlier failures, afterwards confessed that they had been forced to come by their relatives, but never wished to be cured, and had persistently opposed my suggestions.

2. If possible all stimulants ought to be stopped the moment treatment begins. When a patient is taking a bottle or more of spirits a day, this cannot be done without risk of delirium tremens. In such cases, the quantity taken should be rapidly diminished, and, unless the patient is deeply hypnotised, the insomnia, sickness, etc., which are apt to arise should be combated by drugs.

3. It is important, especially at the commencement of the treatment, that the patient should not be left alone. He should always have some trustworthy person near him, to whom he can confide his temptations, and who will aid him in overcoming them.

4. The operator must be persevering and not easily discouraged; patients, who ultimately do well, sometimes relapse more than once during treatment.

5. Even when the craving disappears quickly, the patient ought to be hypnotised regularly

for a month, and afterwards occasionally for a few weeks.

6. As a general rule, the deeper the hypnosis the greater the chance of success. I only know of one instance amongst my own patients where a relapse followed after somnambulism had been obtained. On the other hand, many have recovered when hypnosis had only reached the stage of drowsiness, while in some instances the recovery of the patient was the only evidence of hypnosis.

7. During hypnosis distaste of alcohol ought to be suggested to the patient, as well as the abolition of the craving. Finally, he must be made to understand that he can never look forward to being a moderate drinker, and that the only choice before him lies between total abstinence and drunkenness.

Morphinomania and other Drug Habits.—Morphia and other drug habits frequently yield to hypnotic treatment. In many of my cases of neurasthenia and hysteria, especially where insomnia was a marked symptom, a drug habit had been established. Some took morphia regularly, either by the mouth or hypodermically, while a yet greater proportion used chloral, sulphonal, etc. One patient, who suffered from hysterical neuralgia, frequently inhaled chloroform in addition to injecting morphia. In these cases the disease which had given rise to the drug habit was treated first; if the patients recovered, they often spontaneously gave up the habit or it disappeared in response to further suggestion. Thus, the patient, who had long taken chloroform and morphia, abandoned both as soon as his neuralgia ceased.

A cocaine habit sometimes results from the prescription of cocaine spray for hay asthma or other affections of a like nature. One of my patients, who recovered under hypnotic treatment, had for eight years used an ounce of cocaine per week in this way.

Wetterstrand, amongst other cases of drug habits, cites 38 in which morphia was injected subcutaneously; of these 28 recovered. Many of the cases were extremely grave and of long standing, and with several the abstinence treatment had been tried without success—sometimes more than once. Most of them were treated in private houses; the morphia was never stopped at once, but rapidly decreased, while the patient was constantly informed of the diminution.

Dr. Fulda, Frankfurt-on-Main, reports a case of morphinism of ten years' duration, which was cured after three months' hypnotic treatment; two years later there had been no relapse.

One of Dr. Marot's patients had taken morphia for six years. She could not pass half an hour without giving herself a subcutaneous injection, and her arms, legs, and abdomen were covered with numerous cicatrices and subcutaneous nodules resulting from the use of the syringe. She recovered after hypnotic treatment, and three and a half years later there had been no relapse.

Successful cases are also reported by Voisin, Bérillon, Tanzi, and many others.

Epilepsy.—Elliotson, Esdaile, and Braid reported cases of epilepsy said to have been cured by mesmeric or hypnotic treatment. In one, cited by Elliotson, there had been no relapse after two years, and in one of Braid's after eight years. Both patients had apparently suffered from genuine long-standing attacks of *grand mal.*

Wetterstrand asserts that some of his earlier patients have now remained many years without relapse, and his general results are so startling that Forel said he only accepted them after he had personally investigated Wetterstrand's work.

More or less successful cases are reported by de Jong, Bérillon, Stadelmann, Woods, and others.

With the exception of a case of epilepsy, associated with dipsomania, in which five years have passed without relapse, my results, although encouraging, are not conclusive. Out of eleven cases marked improvement occurred in five instances, but complete recovery in none. In one case of four years' duration, in which there were several attacks of *grand mal* per week, and much impairment of intellect, there was a remission for twelve months; the patient then relapsed, but the fits ceased under renewed treatment. In another somewhat similar case the attacks ceased for eighteen months, then a solitary one appeared, while now twelve months have passed without a further attack.

The cases already cited by no means exhaust the therapeutic applications of hypnotism, and the following are some—amongst the many widely differing diseases—in which it has been employed with success:—

Menstrual Disorders.—According to Braid, hypnotic treatment was the best for regulating menstruation, and for curing or relieving morbid conditions connected with it. Later writers hold similar views. Thus, Forel cites cases in which he personally succeeded in causing the appearance or cessation of the period during hypnosis, and also in regulating its duration and intensity, while, in other instances, he produced the same results by post-hypnotic suggestion.

The following cases are recorded by Tyko Brünnberg, of Upsala:—

	Cases.	No Improvement.	Improved.	Cured.
Amenorrhœa . .	9	1	2	6
Menorrhagia . .	9	3	2	4
Menorrhagia and Dysmenorrhœa	5	1	1	3
Dysmenorrhœa . .	3	1	1	1
Total	26	6	6	14

One of the patients, a girl over 20 years of

age, had never menstruated, but did so after the second hypnosis.

Some of the successful cases of dysmenorrhœa were severe and of long standing, the periods being much lengthened and the intervals shortened.

Grossmann records a case of amenorrhœa, of three years' duration, in a women 30 years of age. A few hours after the first hypnosis, the period commenced slightly, and became copious after the second *séance* next day.

The following case is recorded by Bernheim:—

Madame M., aged 35.—Up to the birth of her first child, menstruation was regular, with an interval of 21 days; afterwards, she suffered from menorrhagia and dysmenorrhœa, the interval between the periods varying from 11 to 13 days. Bernheim suggested a progressive retardation of the periods, a definite time being fixed for the appearance of the menses, *e.g.* 25, 27, 28, and 29 days. These suggestions were successful; an interval of 29 days was established, the dysmenorrhœa ceased, and the duration of the period was reduced from 6 to 3 days.

Successful cases of amenorrhœa, dysmenorrhœa, and menorrhagia are also cited by Delius, Wetterstrand, Voisin, Bérillon, Gascard, Journée, Marandon de Monthyel, Bourru, Burot, Dècle. Many of the cases were of long standing, and had resisted other methods of treatment, while their recovery was confirmed by later reports.

My personal observations accord with the conclusions just cited. In nearly every instance, where deep hypnosis had been induced, the dysmenorrhœa ceased, even when it was associated with various forms of uterine displacement. In many cases of menorrhagia, the length of the interval was increased, and that of the period diminished. Some of these patients, who formerly had to pass at least a week in bed at each period, have now no interruption in the activity of their lives.

In one case of amenorrhœa of eighteen months' duration, the period appeared a few hours after the first *séance*, and again a month later, when hypnosis was reinduced. In another, of eighteen months' duration, the period appeared at the day indicated, in response to a post-hypnotic suggestion made three weeks previously.

In the majority of cases, the hypnotic treatment required was short, and the menstrual functions remained normal after its cessation.

Secretion of Milk. — Several cases are recorded in which the secretion of milk has been increased or arrested by suggestion. One of the earliest is cited by Esdaile. His sister-in-law, when weaning, suffered from an accumulation of milk in her breasts, which rendered them painful and swollen. Esdaile hypnotised her; in half an hour she was free from pain, while next morning the breasts were soft and comfortable, and without further secretion of milk.

An experiment, the reverse of this, was related by Braid. He hypnotised a patient and suggested an increase of milk in one breast. On waking, she had no recollection of what he had said, but complained of tightness in the breast. Her child was fourteen months old, and her milk had almost disappeared; despite this, the breast became almost immediately distended with it. A few days later, Braid successfully repeated the experiment with the other breast. The patient suckled her child for six months longer, the supply of milk being more abundant than it had been at any time since her confinement.

The following case is reported by Grossmann: —B., aged 20, primipara, suckled her child for a fortnight; then ceased to do so as she had to leave home. Three weeks later, she returned and wished to nurse the child again, but she had no milk in the right breast and hardly any in the left. The patient was hypnotised, and the sensations associated with the flow of milk suggested. In three minutes, the veins of the left breast became enormously congested, and milk began to flow from it. At first, repeated pressure failed to produce a single drop from the right breast, but when the suggestions were repeated it was secreted freely.

An interesting case is recorded by Freud, where a patient was unable to suckle her child, not from want of milk, but because violent vomiting followed whenever the child was put to the breast. This symptom had appeared after a previous confinement, and the patient had been compelled to abandon her attempts to suckle. On the present occasion, and also on a subsequent one, hypnotic suggestion rapidly removed all difficulty.

Stammering.—As a rule, good results depend upon deep hypnosis, but exceptions occur, and cure may take place when slight hypnosis alone has been induced. Thus, one of my patients, an inveterate stammerer, whose infirmity rendered him self-conscious, shy, and miserable, completely recovered although he barely reached the stage of lethargy. All the other morbid symptoms disappeared when the stammering ceased, and the patient has recently been called to the bar. Wetterstrand reports 48 cases, average age fifteen years, with the following results: recovered 15, improved 19, no improvement 14.

Sea-Sickness.—Slight hypnosis has apparently little effect in preventing sea-sickness. The results, however, are often very successful when profound hypnosis can be induced. Here, even when the suggestions have been made months previously, sickness is often entirely prevented, even with patients who had previously been invariably ill on short and calm voyages. One of my patients, who before treatment had been

a phenomenally bad sailor, afterwards made several voyages to and from India, without sickness or rehypnotisation.

Rheumatism.—Grossmann records 123 cases of chronic articular rheumatism, and claims to have obtained good results in every instance. Tatzel, Delius, and others, also report successful cases. As far as my personal experience goes, the pain of all forms of rheumatic affections, both acute and chronic, can be relieved or abolished by suggestion in deep hypnosis. In one instance, a commencing attack of rheumatic fever was apparently aborted by keeping the patient in the hypnotic trance for forty-eight hours. In two cases of rheumatoid arthritis good results were obtained, even although slight hypnosis alone was induced, *e.g.* Mr. ——, aged 45. Illness of two years' duration. Joints swollen and distorted; walked badly, use of hands and feet much impaired, and suffered from insomnia and severe pain. Careful treatment at home and at foreign baths had produced no improvement. After hypnotic treatment all morbid symptoms, except those due to organic changes in the joints, disappeared and the patient resumed work.

Miss ——, aged 26.—Illness of seven years' duration. All the joints of the extremities were affected, knees ankylosed, elbows and wrists almost immovable. Patient unable to walk, or even to move in bed; she invariably slept on her back, and could not turn on to her side. The stiffness in the hip joints prevented her sitting; she was in constant pain, and suffered from emaciation and amenorrhœa. Result of hypnotic treatment: Patient now strong and well nourished. She sleeps well, is free from pain, and menstruation is regular. The absence of pain enabled passive movements to be made, and now, although there is still much stiffness and distortion, the patient can move freely in bed, walk with crutches, sit up with comfort, and use her hands for various forms of work.

Deafness.—The range of hearing can be increased in the healthy individual by suggestion; many instances are also reported in which improvement or recovery has taken place in cases where the hearing had been more or less impaired by accident or disease. In the majority of those I have observed, there was a history of scarlet fever with otorrhœa. In most cases the perforation of the drum had healed, and there was little to be observed in the way of physical abnormality. The hearing, however, was defective on one or both sides, the watch being heard sometimes only at a distance of an inch or two, at others only on pressure. When deep hypnosis was induced, the results were frequently striking, and the hearing became normal or even hyperæsthetic. Several years later the improvement was still maintained.

In cases of total deafness—congenital or otherwise—no improvement was obtained.

In Menier's disease the results are interesting. In a typical case, there had been sixteen years' deafness of the right ear and two years of the left. At first, the patient had suffered from occasional attacks of headache, giddiness, etc., these being followed by an increase in deafness. Later, there was marked vertigo, the patient was unable to walk unaided, and this was associated with rotatory movements, constant headaches, and numbness of the hands and feet. In all cases where deep hypnosis was induced, there was a slight improvement in hearing, with partial or complete disappearance of all the nervous symptoms. A year later, these patients reported that the improvement in hearing had not been maintained, but that there had been no return of the nervous symptoms.

Skin Diseases.—The following cases are from my own practice:—

Mrs. ——, aged 49.—Pruritus vulvæ and eczema of four years' duration, unrelieved by treatment. Intense nocturnal irritation, insomnia, and chronic constipation. The uterus was retroflected and bound down by adhesions; this, together with the constipation, was supposed to have originated and maintained the disease. The sphincter ani was stretched under ether by Mayo Robson, but this neither cured the constipation nor relieved the other symptoms. Hypnosis was induced at the sixty-seventh attempt. Result: Rapid and complete recovery, with no relapse after three years.

Miss ——, aged 15.—On the back of the left arm, just above the wrist, a patch of skin, $2\frac{1}{2}$ inches long by $1\frac{1}{2}$ broad, was the seat of constant perspiration, which had existed from early childhood. This was always excessive, saturating the bandages which enveloped the arm, and dripping upon the floor. Hypnosis, with somnambulism, was induced at the first attempt, and next day the perspiration had markedly diminished. The patient was again hypnotised, when the perspiration ceased, and two years later there had been no relapse.

Successful cases are reported by other writers. Thus, Hamilton Osgood cites (1) a case of severe and intractable eczema of nine years' duration in a boy, aged eleven. Hypnotised. Recovered. (2) Mrs. ——, aged 88, severe eczema of nine years' duration. Hypnotised. Recovered. (3) Mr. ——, aged 60, severe and painful eczema of forty years' duration. Hypnotised. Recovered.

Therapeutic Summary.—*Insanity.*—Forel, while admitting the genuineness of Voisin's therapeutic results, asserted that his so-called insane patients simply suffered from hysteria. The brain, he says, is the instrument we employ in suggestion, and if the instrument itself is spoilt, we are no longer able to make use of it, or only to a very small extent, as a means of reacting upon itself and influencing its functions in general.

In reply to this, Voisin, while denying that

all his cases were hysterical, claimed that the successful treatment by suggestion of that class alone, marked a therapeutic advance of no little importance. Our asylums, he said, contained a large number of hysterical insane, who were violent and dangerous, and who inspired both disgust and pity by their tendency to drink and steal, and by their lies, dirtiness, obscene and unnatural acts. Many of those, who had long been asylum inmates, were, thanks to hypnotism, now leading active and useful lives.

Later, Forel himself reported a case of delusional insanity, which he had successfully treated by hypnotism. The question whether insanity in its graver forms can be cured by hypnotism is still an open one, but one must not forget, in endeavouring to estimate the value of this form of treatment, that the experiment practically dates from Voisin's first case in 1880, and that comparatively few attempts have been made by others to give the method a fair trial. The value of the Bethlem cases is much lessened by the fact that in no instance did the number of attempts to induce hypnosis exceed nine, while in six cases only one was made.

A certain proportion of the insane are cured or recover, and it seems reasonable to suppose that a method of treatment which, without drugs, can produce sleep, remove excitement and other morbid mental conditions, may prove a valuable remedial agent.

Epilepsy.—Here, the results are encouraging, but not conclusive. In some instances, hystero-epilepsy may have been mistaken for genuine epilepsy, while in others, sufficient time has not elapsed to enable us to judge of the permanency of the cure. On the other hand, if other medical men have not obtained successes equal to those claimed by Wetterstrand, few, if any of them, have employed his special method, viz. prolonged hypnotic sleep.

Other Diseases.—Apart from insanity and epilepsy, the therapeutic results must be regarded as satisfactory, and the following are some of the more important points as to the statistics on which these results are based :—

1. The cases observed have been numerous. Thus, to take a few examples, van Eeden and van Renterghem treated 1089 persons between the years 1887 and 1893, while Wetterstrand's cases up to 1893 amounted to 6500. Liébeault has published no complete record of his thirty years' practice, but, from 1887 to 1890, he treated 1756 cases. From 1882 to 1886, Bernheim attempted to hypnotise 5000 of his hospital patients, while a few years later the number had risen to 10,000. In the first year of his hypnotic practice, Forel treated 205 patients, many of whom were insane. In 1898, he informed me that he had tried to hypnotise about 1000 persons, and had succeeded with over 95 per cent.

2. Many of the observers, notably Forel, had already gained distinction not only in medicine, but in other departments of science.

3. The majority of the observations were checked by others. Thus, Bernheim's cases were watched by Beaunis and Liégeois, while the majority of Voisin's were hospital patients, who were under the observation of keenly critical and sometimes hostile colleagues. Further, the cliniques, even when these were not hospital ones, were always open to other medical men, and I have spent many weeks amongst the patients of Liébeault, Forel, Wetterstrand, van Eeden, van Renterghem, Bernheim, etc.

In the therapeutic section of this article successful cases are alone cited. Naturally, however, hypnotism, like other methods, has its failures. These are due to many causes, amongst which the following may be cited :—(1) The selection of unsuitable cases. Organic diseases, which do not respond to other forms of treatment, are equally incurable by hypnotism. In such maladies, however, some of the symptoms are due to functional disturbances, excited beyond the actual limits of organic lesion, and these are often relieved by hypnotism. (2) Hypnotism is rarely resorted to until all other forms of treatment have failed. The disease is thus engrained, as it were, and the patients have the fixed idea that they cannot be cured. Recovery, under such circumstances, gives a special significance to hypnotic statistics. (3) The time allowed is often far too short. Patients who for twenty years have tried other methods in vain, grudge giving a few months to hypnotism, while others regard the treatment as a sort of operation, which they condemn as unsuccessful if it does not cure at the first sitting.

In judging hypnotic results, therefore, there are three things which ought specially to be remembered. (*a*) The cases treated in this country are almost invariably those which have been unrelieved by other methods. (*b*) Even under these unfavourable circumstances, the percentage of success is high, and many functional nervous disorders, which apparently receive little or no benefit from drugs, are relieved or cured by hypnotism. (*c*) Further, with due care hypnotic treatment is absolutely devoid of danger. The will is strengthened instead of weakened, and the patients soon learn to rely upon themselves instead of upon the operator.

Hypnotism in Surgery

Esdaile justly claimed that painless mesmeric surgery was common in his hospitals, years before the days of ether and chloroform.

The following cases from my own practice illustrate hypnotism as an anæsthetic :—Miss A., aged 20.—Double strabismus. Operated on by Bendelack Hewetson, 1889. Hypnotic anæsthesia alone. The patient turned her eyes as directed, so as to put the muscular fibres on the stretch. Later, she sustained a severe fracture of the

nose. Hypnosis was induced and the bones moulded into position. There was no pain during or after these operations.

Mr. B., aged 40, 1890.—Fracture of bones of arm and shoulder girdle, with injury to soft parts and subsequent ankylosis of joints. The adhesions were frequently broken down under chloroform. Result, swelling, inflammation, and renewed immobility. Ultimately, the patient refused the anæsthetic, and would not allow passive movements without it. He was hypnotised at the first attempt, and the adhesions broken down without pain; this was repeated from time to time, and followed by complete recovery.

The late Mr. A. Turner, L.D.S., published an account in the *Dental Journal* for March 1890, of some of his operations on my patients. He stated that on one occasion he had extracted forty teeth, selecting those cases likely to afford a severe test. Hypnosis was induced and terminated almost instantaneously; anæsthesia was complete, and yet the movements of the patient could be controlled by the operator, thus obviating the necessity for gags, etc. A weak, anæmic girl, with valvular disease,—likely to "deepen" under nitrous oxide, or to collapse for a day after ether,—was quickly and quietly rendered unconscious. Two left and two right molars, and a lower bicuspid, all difficult teeth, were painlessly extracted and without subsequent suffering.

On 28th March 1890, a number of my patients were operated on at Leeds, and, according to Mr. Pridgin Teale, who was present with sixty other medical men, "the experiments were deeply interesting, and had been marvellously successful."

The following were two of the cases:—

C., aged 19.—Dental caries. A weak, anæmic girl. Hypnotic anæsthesia was produced by my written suggestion, and sixteen teeth extracted, while I remained in another room.

D., a boy aged 8.—Exostosis of great toe. Hypnosis first induced two days previously. Mr. Mayo Robson removed the great toe nail, then the exostosis, and part of the first phalanx.

In none of the cases was there any pain either during or after operation. Some were nervous subjects, highly sensitive to pain in the normal condition; others were strong, healthy working men. All returned to Goole the same day, and in every case the healing process was remarkably quick.

Further operations are reported by other observers, thus:—Tillaux, colporrhaphy; Schmeltz, ectropion and amputation of breast; Bourdon, uterine fibroid; Grossmann, fractures and dislocations; Sandberg, dental operations; Forel, cataract; Wood, necrosis of humerus; van Eeden and van Renterghem, dental operations.

Granting, as recent statistics apparently show, that 94 per cent of mankind can be hypnotised, it must still be admitted that many preliminary attempts are required in a large proportion of cases. Further, hypnosis frequently never becomes deep enough for operative purposes; this is specially noticeable amongst the nervous and hysterical. Under these circumstances, unless there were grave objections to the use of other anæsthetics, I should consider it a waste of time to attempt to hypnotise a patient for operative purposes alone.

Apart from this, hypnotic anæsthesia possesses many advantages, thus:—(1) Once deep hypnosis, with anæsthesia, has been obtained, it can be immediately reinduced at any time; (2) No repetition of any hypnotic process is necessary, the hypnotiser's written or verbal command is sufficient; (3) The hypnotiser's presence is not essential, the patient can be put in touch with the operator by written order, or by other means previously suggested during hypnosis; (4) No abstinence from food or other preparation is necessary; (5) Nervous apprehension can be removed by suggestion; (6) Hypnosis is pleasant and absolutely devoid of danger; (7) Hypnosis can be maintained indefinitely and terminated immediately at will; (8) The patient can be placed in any position without risk—a not unimportant point in operations on the mouth and throat—and will alter that position at the command of the operator; (9) Gags and other retentive apparatus are unnecessary; (10) Analgesia alone can be suggested, and the patient left sensitive to other impressions—an advantage in operations on the mouth and throat; (11) There is no tendency to sickness during or after operation, a distinct gain in abdominal cases; (12) Pain after operation, and during subsequent dressings, can be entirely prevented by suggestion; (13) The rapidity of the healing process, possibly as a result of the absence of pain, is frequently very marked.

Hypnotism in Obstetrics

Elliotson and other mesmerists of his day reported many cases of painless confinement, and in their time newspaper announcements of births were sometimes followed by the words "painlessly during hypnotic trance." Many instances of painless hypnotic birth are reported by modern observers; amongst others, the following cite cases in which all the patients were primiparæ—Kingsbury, Schrenck-Notzing, Wetterstrand, Mesnet, Fraipont, Fanton, Dobrolsky.

Dr. Bourdon suggested to a patient, in labour at the full term, that the pains should cease and not reappear for eight days. He claims that the experiment was successful, and that the confinement took place painlessly at the time he had selected.

Schrenck-Notzing states that hypnotic suggestion can render labour painless. It also facilitates its course by regulating the position of the body and limbs, and increasing or diminish-

ing the uterine contractions through the action of the voluntary muscles.

Dr. Fanton asserts that suggestion acts directly upon the uterus, and that the operator can cause its contractions to appear or disappear at will. Thus, uterine inertia may be successfully combated without ergot, and the use of forceps frequently rendered unnecessary. The after-contraction of the uterus can be excited and maintained by suggestion, and post-partum hæmorrhage prevented; while, in cases of adherent placenta, turning, etc., the uterus can be relaxed and the necessary operations facilitated. Dr. Fanton claims to have excited premature labour by suggestion, and considers it criminal to have recourse to other means—dilatation of the cervix, perforation of the membranes, etc.—without having first attempted this method.

Although Dr. Fanton founds his statements on numerous cases and experiments, many of them observed and confirmed by other medical men, the results he claims are so startling that few are likely to accept them without further evidence. One must admit, however, that the involuntary contractions of the uterus are sometimes arrested by emotional states. Thus, when a patient is in active labour, her pains may suddenly cease and remain in abeyance for hours, if a stranger comes to attend her instead of her own medical man.

Pregnant women are more easily hypnotised than those suffering from nervous diseases; and from this, and other reasons, there are fewer objections to the employment of hypnotism in obstetrics than in general surgery. Notwithstanding, unless the methods of inducing hypnosis are greatly improved, its use as an anæsthetic will ever remain restricted. Hypnotic anæsthesia, however, must always be of keen interest to both physiologist and psychologist. It is a thing apart, not an ordinary narcotic, illustrating familiar methods of preventing pain by checking all conscious cerebration. It is a new departure—the first successful attempt at dissociating forms of sensation, which throughout the known history of the human organism have almost invariably existed together. Here, pain is suppressed by the inhibition, from among all the subject's possible sensations, of precisely those which would in any way be disagreeable to him, while general consciousness may be unimpaired, and sensations, other than painful ones, rendered more acute by suggestion.

Stages

The hypnotic condition is divided by different observers into a varying number of stages. Most of these classifications, particularly Bernheim's, are complicated and misleading, while none are quite satisfactory, as the stages themselves are more or less the result of training, and thus artificial.

The following classification is the one I follow:—

1. *Slight Hypnosis.*—Changes in the voluntary muscles can be induced. Memory unaltered.
2. *Deep Hypnosis.*—Here, in addition, changes in the special senses can be evoked, and there is sometimes partial amnesia on waking.
3. *Somnambulism.*—In this stage, all the phenomena of the two previous ones can be evoked, and sometimes also others (appreciation of time, etc.), which cannot be produced in the earlier stages. The chief characteristic of somnambulism, however, is the amnesia which follows it, *i.e.* the waking consciousness retains no recollection of what has occurred in hypnosis.

The division of the hypnotic state into different stages is of little practical importance to the physician. With rare exceptions—such as the production of somnambulism for operative purposes—none but curative suggestions are given, and, in the majority of the successful cases, recovery takes place before any deep stage is reached. With a certain proportion of such patients, the phenomena of deep hypnosis might be evoked and classified, if suggestions were directed to that end. Experimental and therapeutic work, however, should be kept absolutely distinct.

Susceptibility

In 1892, Schrenck-Notzing published his *First International Statistics of Susceptibility*, which showed that fifteen observers in different countries had tried to hypnotise 8705 persons, with the following results:—6 per cent were uninfluenced; 15 per cent became somnambules, while 78 per cent were less deeply hypnotised. These figures are fairly representative, although certain observers have obtained still better results. Thus, out of 1756 consecutive cases, Liébeault's failures only amounted to 3 per cent, while Wetterstrand only failed in 3·7 per cent out of 3209 cases.

Susceptibility is little affected by race, and hardly at all by sex; but children are more easily influenced than adults. According to Forel, every mentally healthy person is naturally hypnotisable, while the majority of observers agree that the hysterical and ill-balanced are the most difficult to influence.

The causes which affect susceptibility adversely are too numerous to be referred to in detail; the most important are fear and disquieting emotions of a like nature. Thus, the psychical preparation of a patient for hypnosis is as important and complicated as the physical preparation essential to antiseptic surgery.

In the first 500 cases, drawn from my general practice, there were only two failures, while 48 per cent became somnambules. This susceptibility, far above the average, was apparently due to the following causes:—I had already gained the patients' confidence by attending

them in previous illnesses. At that time hypnotism was not discussed in the public press, and the patients had not been frightened with absurd stories as to its dangers. Many of the patients were healthy persons hypnotised for operative purposes, while the percentage of cases of long-standing nervous disease was small.

In the first 100 cases, drawn from other sources, the failures reached 22 per cent. Here, most of the patients suffered from serious and long-standing nervous disease. All were strangers to me, while the majority had heard that hypnotism was dangerous and inevitably associated with loss of consciousness and volition.

Remarks on Methods and the Management of the Hypnotic State

Some further hints, as to the induction of hypnosis and the management of the hypnotic state, may be useful to those about to practise hypnotism.

1. Do not attempt to hypnotise your patient at the first interview. Hear his history; form your diagnosis, and make friends with him. Then tell him that what you propose doing involves neither interference with volition nor possibility of harm. Explain that he will have to rest quietly, with his mind fixed on some monotonous idea, while you make suggestions, and that these, when given under specific circumstances, tend to arouse powers which are latent in his own brain, and which may be used for his benefit. Above all, emphasise the fact that only a very small proportion of those who are cured pass into a condition in any way resembling sleep.

2. At the next visit, place the patient in a comfortable chair and darken the room. Then, after making him gaze fixedly upwards for a few seconds, tell him to close his eyes, and to keep them voluntarily shut until you request him to open them. Meanwhile, verbally suggest that he shall pass into a more restful and drowsy state each time he visits you, and lose all his morbid symptoms. The latter suggestions must be varied according to the nature of the case; you must also explain to the patient that they are not likely to be realised at once, and that frequent repetitions are often necessary.

Sometimes patients are easily hypnotised at the first attempt, and respond at once to curative suggestions. With the majority, however, the length of treatment required bears some proportion to the duration of the disease and the mental instability of the patient, and I have sometimes only succeeded in inducing hypnosis after a hundred failures.

Curative suggestions, repeated in a more or less varied form, are usually given for ten minutes to half an hour at a time. According to Wetterstrand, however, too much importance has been attached to suggestion, while the health-giving action of hypnotic sleep has not been sufficiently recognised. He records many cases of hysteria, epilepsy, morphinomania, etc., where the patients were kept in the hypnotic state for a month or more. Nurses, put *en rapport* with them, fed them at stated intervals, while the action of the bladder and bowels was regulated by suggestion. The results were extremely satisfactory, and no untoward symptoms of any kind occurred.

This method is only applicable to cases of deep hypnosis and somnambulism; fortunately, however, the majority of subjects respond to curative suggestions as soon as the slightest hypnosis has been induced, and thus Wetterstrand's method of prolonged sleep is rarely necessary. In some instances, however, it succeeds when ordinary hypnotic methods have failed. Thus, one of my patients, who had suffered from insomnia, spinal neuralgia, dysmenorrhœa, and menorrhagia, was cured of the three former conditions by suggestion. The fourth was unrelieved, and the patient had to remain in bed a full week out of each month. She was kept in the hypnotic trance for three days before a period, and then for five during it, while frequent suggestions were given. Since then the interval, duration of the period, and amount of loss, have become normal, and the patient, instead of remaining in bed, can now take active exercise without inconvenience.

Hypnotic Theories

First, to summarise the phenomena requiring explanation: The somnambule recalls the events of normal as well as of hypnotic life, but, when hypnosis is terminated, remembers those of ordinary life alone. Further, he can influence and control his own mind and body to an extent undreamt of by the waking will. Finally, there is no impairment of consciousness or volition, nor, in proper hands, the slightest danger.

In slight hypnosis memory is unchanged, but an increased power of controlling the organism always exists.

(*a*) *The Mesmeric Theory.*—According to the mesmerists, the phenomena were due to a vital fluid or physical force, which was transmitted from operator to subject. This force also existed in certain metals, crystals, and magnets, but all were not influenced by it; those who could be were termed "sensitives."

(*b*) *Braid's Theories.*—The subjective nature of the phenomena—the fact that they were due to changes in the patient's own brain—was the essential part of all Braid's theories. On other points, his views changed with increased knowledge. At one time, he attributed everything to an involuntary monoideism; this view was published by John Hughes Bennett in 1851. Here, the phenomena were explained physiologically by the arrested action of some of the "fibres of association." Psychologically by dominant ideas; these had obtained undue

influence, because other ideas, which ought to have controlled them, did not arise, as the portion of the brain, with which the latter were associated, had its action temporarily suspended, *i.e.* the connection between the ganglion cells was broken, owing to the interrupted action of the fibres of association.

Later, Braid believed that volition was unchanged in hypnosis, the moral sense increased, and suggested crime impossible. He observed that several phenomena could be evoked simultaneously, and without the subject having passed through any condition resembling sleep. Verbal suggestion excited, but did not explain, the phenomena; the healthy were the easiest to influence, and the condition, as a whole, could only be accounted for by the intelligent action of a secondary consciousness.

(*c*) *The Salpêtrière Theory.* — This, in all essential points, reproduces that of the mesmerists. The old fallacies as to metals, magnets, drugs in sealed tubes, etc., are revived; the subjects, however, instead of being termed "sensitives," are called "hysterical."

(*d*) *Theories of the Nancy School.*—Bernheim reproduces the mental side of Bennett's theory, and ascribes the phenomena to a psychological inhibition. In addition to adopting Braid's discarded view, Bernheim reproduces some mesmeric errors, and adds others of his own. He believes in *rapport*, the possibility of suggesting crime, and the practical identity of hypnosis with normal waking and sleeping life. There is, he says, no special state deserving the name of hypnosis, *suggestion* is everything.

In some recent theories, claiming originality, the physiological half of Bennett's masquerades in modern terminology. Thus, instead of "association fibres" we have "neurons" and "neuronic groups"; "dendrons" contract and break the contact between nerve cells, while a supposed decrease in cerebral activity is explained by an "inhibition of the amœboid movements in the pseudopodic protoplasmic prolongations of the neuro-spongium."

(*e*) *The Subliminal or Secondary Consciousness Theory.*—This theory presupposes an intelligent secondary consciousness, and explains the phenomena of hypnotism by the arousing of powers over which we normally have little or no control, instead of by the arrested action of some of the brain centres which subserve normal life. As William James points out, the principle on which it depends is largely admitted by science, and it gives, what no other theory does, a convenient working hypothesis. The characteristic phenomena of hypnosis show increased, not diminished, mental power. Cerebral inhibitions, no matter how involuntary or incomplete, do not explain improvements in memory, arithmetic, and appreciation of time, and the subliminal consciousness theory marks an important advance towards solving these problems.

Hypo-.—In compound words *hypo-* (Gr. ὑπό, under) has the significance of defective, and is opposed to *hyper*.

Hypoagnathus. — The teratological type in which there is absence of the lower jaw (often in association with cyclopia); agnathus.

Hypoalbuminosis. — Deficiency in the albumin of the blood.

Hypoazoturia. — Deficiency in the amount of nitrogen in the urine.

Hypoblast.—The innermost of the three layers of the blastoderm; the entoderm *See* EMBRYOLOGY; FŒTUS AND OVUM, DEVELOPMENT OF; etc.

Hypobulia.—Morbid weakness in exercising the will; it is opposed to hyperbulia (*q.v.*).

Hypocatharsis.—A slight degree of purging.

Hypocaustum. *See* BALNEOLOGY (*Historical*).

Hypochlorhydria. — Deficiency of hydrochloric acid in the gastric juice.

Hypochondriac Region. *See* ABDOMINAL TUMOURS, DIAGNOSIS OF; GYNÆCOLOGY, DIAGNOSIS IN (*Physical Examination, Abdomen*).

Hypochondriasis. *See* HYSTERIA, SURGICAL ASPECTS OF (*Resemblance to Hypochondriasis*); PROSTATE GLAND (*Chronic Prostatitis, Sexual Hypochondriasis in*); SENILE INSANITY (*Psychoses of Senility*); STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology, Nervous, Hypochondriasis*); THERAPEUTICS, HEALTH RESORTS (*Reasons for sending Patients Abroad*); URETHRA, DISEASES OF (*Stricture, Symptoms*).

Hypoclysis.—The administration of an enema or clyster (Gr. ὑποκλύζω, to wash from below).

Hypodermic.—Subcutaneous, especially in relation to the administration of drugs. *See* ASEPTIC TREATMENT (*Disinfection of Hypodermic Syringes and Solutions*); PLEURA, DISEASES OF (*Pleurisy, Differential Diagnosis*); SYPHILIS (*Treatment, Hypodermic Injection of Mercury*).

Hypodermoclysis.—The injection of serums or of saline solution (usually in considerable amount) into the subcutaneous tissue.

Hypoepinephry. — A syndrome in which there is functional insufficiency of the adrenal glands; there are asthenia, arterial hypotension, peripheral vaso-motor disturbances, and various nervous and digestive troubles; sudden death is not uncommon; it is to be distinguished from Addison's disease, in which there is melanoderma.

Hypogastric Region. *See* Abdominal Tumours, Diagnosis of; Gynæcology, Diagnosis in (*Physical Examination, Abdomen*).

Hypogastroschisis. — The teratological type in which there is defect of the anterior abdominal wall in the hypogastric region, with protrusion of the viscera.

Hypogeusia.—Defective sense of taste; is opposed to *hypergeusia.*

Hypoglossal Nerve, The.

See also Brain, Tumours of (*Localising Symptoms*); Brain, Cerebellum, Affections of (*Tumour, Symptomatology*); Tabes Dorsalis (*Symptomatology, Affections of Cranial Nerves*).

The hypoglossal or twelfth cranial nerve is the homologue of the anterior root of a spinal nerve. It is interesting to note that in some animals the twelfth nerve possesses a posterior root furnished with a ganglion like a spinal nerve. A similar condition has been met with in a few instances in man (Quain).

The nerve arises from a nucleus which is the direct continuation in the medulla of the multipolar cell columns of the anterior horns of the spinal cord. This nucleus, which lies close to the middle line, in its lower part is situated anterior and external to the central canal. Higher up it comes to lie on the floor of the fourth ventricle, internal to the nuclei of the glosso-pharyngeal, vagus and spinal accessory (see vol. i. p. 472). The nerve fibres leave the nucleus in successive bundles, which run obliquely forwards through the substance of the medulla. They lie external to the fillet and pyramid, and eventually appear in the groove between the pyramid and olive, on the anterior surface of the medulla. Ten to twelve of these bundles unite to form the hypoglossal trunk which passes through the anterior condyloid foramen.

After leaving the cranium the nerve descends almost vertically to the lower border of the digastric muscle, lying between the internal carotid artery and the jugular vein. It is then directed forwards above the hyoid bone, and passes beneath the digastric, the lower end of the stylo-hyoid and the mylo-hyoid muscles, while it rests upon the hyoglossus. Finally, by piercing the genio-glossus muscle it reaches the tongue, to which its terminal branches are distributed.

The hypoglossal, which is purely a motor nerve, has, according to Quain, the following distribution: "The hypoglossal nerve proper supplies only the muscles of the tongue, with the exception of the palato-glossus and the pharyngeo-glossus. Fibres derived from the first three cervical nerves, which are associated with the hypoglossal for a part of their course, are distributed to the infrahyoid muscles and the genio-hyoid, others of uncertain origin pass to the skull and dura mater and to the internal jugular vein. The hypoglossal forms connections with the pneumogastric, lingual, upper three cervical nerves, and sympathetic."

The hypoglossal nerve is the motor nerve of the tongue, of the depressors of the hyoid bone, and of some of the elevators. Since the nerve is in great part distributed to the tongue, it is in that organ that the more important effects of lesions of the nerve are to be looked for.

When one hypoglossal nerve is paralysed the tongue, when protruded, deviates to the paralysed side. If there is complete bilateral paralysis, the tongue lies on the floor of the mouth and protrusion is impossible. Articulation, mastication, and to some extent deglutition, are interfered with when the tongue is completely paralysed. On the contrary, if the lesion be unilateral there may be little or no impairment of these functions. If a lesion involves the nerve or its nucleus, the tongue wastes and the mucous membrane forms characteristic furrows on its surface, while corresponding diminution occurs in its electrical excitability. Under these circumstances fibrillary tremors are frequently to be seen in the wasted muscle. When the supranuclear portion of the nerve is the seat of the lesion these changes do not occur.

The upper hypoglossal neurons, the nucleus, or the nerve in any part of its course, may be damaged by disease. Outside the cranium the nerve may be implicated in a new growth. Occasionally it is divided in an attempt to commit suicide. A neuritis of the hypoglossal nerve is of very rare occurrence, possibly because the nerve lies deeply and is not exposed. A few cases of supposed rheumatic neuritis have been recorded. In its course within the skull the nerve may be involved by meningitis, tubercular and syphilitic growths, disease of the bone, or neoplasms. Paralysis of one-half of the tongue alone rarely results from a lesion in this situation; other nerves are almost always involved. Within the medulla the nuclei or nerve fibres may be damaged by a tumour, hæmorrhage, local inflammation or softening, when the resulting paralysis is usually bilateral. Degenerative lesions affecting the hypoglossal together with adjacent motor nuclei occur in bulbar palsy.

The comparatively frequent association of hæmiatrophy of the tongue with paralysis of the palate and vocal cord on the same side in central lesions suggests a common origin for the motor supply of these structures. Weakness of the orbicularis oris is often associated with paralysis of the tongue. In hemiplegia unilateral paralysis of the tongue is common; in pseudo-bulbar paralysis, a bilateral palsy more

or less complete, and due to a supranuclear lesion, may be met with.

The diagnosis of the situation of the lesion can often be arrived at by a consideration of the associated paralyses.

The treatment must be directed towards the primary disease.

Hypognathus. — A type of double monster in which a more or less defective parasitic fœtus is attached to the lower jaw of the antosite.

Hypolepsis. — Monomania (Gr. ὑπό, under, and ληψις, taking hold of).

Hypoleucocytosis. — Diminution in the number of leucocytes, as in typhoid fever; leucopenia.

Hypomania. — A subacute form of mania, characterised by egotism, intolerance of correction, restlessness, and perversion of sexual instincts.

Hypometropia.—Myopia.

Hypophosphites. *See also* Calcium; Pharmacology; Sodium; etc. The hypophosphites of calcium and sodium (official), and also those of potassium, iron, and ammonia, are much used as general and nerve tonics in debility, incipient phthisis, convalescence from acute illness, etc. They are contained in a large number of proprietary preparations made up with syrup, glycerine, or malt, and containing in addition strychnine, quinine, formates, or other tonic drugs. Calcium hypophosphite has been recommended for checking the night-sweats of phthisis, and also for acne. The dose of the hypophosphites is from 3 to 10 grains.

Hypophysis Cerebri.—The pituitary body. *See* Pituitary Body.

Hypoplasia. — Defective formation or arrested development of a tissue or part.

Hypopyon.—The presence of pus in the anterior chamber of the eye. *See* Iris and Ciliary Bodies (*Inflammatory Conditions, Iritis*); *see also* Choroid, Diseases of (*Suppurative Choroiditis*); Cornea (*Inflammation, Ulcerative Keratitis*); Cornea (*Types of Corneal Ulceration*).

Hypospadias.—Defective development of the urethra on its inferior aspect, so that the canal ends on the lower surface of the penis or in the perineum. *See* Hermaphroditism (*Male Pseudo-Hermaphroditism*); Scrotum and Testicle, Diseases of the (*Abnormalities, Cleft Scrotum*); Scrotum and Testicle, Diseases of (*Impotence, Causes*); Urethra, Diseases of (*Abnormalities*); Uterus, Malformations of the (*Arrest of Development*).

Hypostasis. — The gravitation of the blood to the most dependent parts of the body, *e.g.* after death; venous hyperæmia from the action of gravity; post-mortem lividity. *See* Death, Signs of; Lungs, Vascular Disorders of (*Passive Congestion, Hypostatic*); Medicine, Forensic (*Post-mortem Examinations, External Appearances*).

Hypostomus.—The teratological type in which a small vertical slit represents the mouth, *e.g.* in cases of agnathus or absence of the lower jaw.

Hypothenar Eminence. — The eminence or projection on the palm of the hand on the ulnar side, as opposed to the thenar eminence on the radial side.

Hypothermia. — A low temperature, *e.g.* in the remissions of some fevers (typhoid), or after hæmorrhage, or during convalescence.

Hypo-thyroidea.—Defective action of the thyroid gland and the effects resulting from it. *See* Thyroid Gland, Medical (*Myxœdema*).

Hypotonia or **Hypotonus.** *See* Spinal Cord, Medical (*General Symptomatology, Motor Symptoms, Hypotonia*); Tabes Dorsalis (*Symptomatology, Muscular Tonus*); Tendon-Jerks (*Indications from, Diminished Muscular Tonus*).

Hypoxanthin. — Oxypurin, a leucomaine ($C_5H_4N_4O$) found normally in various tissues (spleen, brain, thymus, marrow, adrenal gland, etc.), and occasionally in blood and urine and in the liver tissue. *See* Liver, Physiology of (*Regulation of Supply of Proteids*); Physiology, Excretion (*Urine, Nitrogenous Substances*); Uric Acid.

Hypsonosus. — A morbid condition affecting mountain climbers, characterised by sickness, headache, and distension of the superficial veins.

Hypsophobia.—Morbid fear of being on heights.

Hysteralgia.—Pain in the uterus.

Hysterectomy.—The operation for the removal of the uterus (in whole, panhysterectomy, or in part, sub-total hysterectomy) either by the abdominal or the vaginal route, for various morbid states, but chiefly for fibroids and cancer. *See* Uterus, Malignant Tumours of (*Treatment, Hysterectomy*); *see also* Labour, Injuries to Generative Organs (*Rupture of Uterus, Treatment*); Menstruation and its Disorders (*Amenorrhœa after Hysterectomy*); Pelvis, Perineum and Pelvic Floor (*Prolapsus Uteri, Treatment*); Pregnancy, Pathology, Affections of Generative Organs (*Cancer of Uterus, Treatment*).

Hysterexopexy. — An operation by which the uterus, or more often a tumour growing from it, is drawn through an abdominal incision and fixed there in an extraperitoneal position; later, the tumour may be removed.

Hysteria. — This is described in the following sections:—

1. **General Article on Hysteria.**
2. **Hysteria in Childhood.**
3. **Surgical Aspects of Hysteria.**

General Article on Hysteria

See also Alcoholism (*Complications*); Amblyopia (*Functional, Hysterical Amblyopia*); Ankle-Joint, Region of, Diseases (*Hysterical Disease*); Asthenopia (*Neurasthenic*); Athetosis (*Diagnosis*); Auditory Nerve and Labyrinth (*Nerve-Deafness in Hysteria*); Brachial Plexus, Surgical Affections of (*Tears and Contusions, Diagnosis*); Brain, Tumours of (*Diagnosis*); Catalepsy (*Etiology*); Cautery (*Black Heat*); Chest-Wall, Affections of (*Pain in the Chest*); Chorea (*Symptoms*); Colour Vision (*Acquired, in Hysteria*); Deformities (*Club Foot, Hysterical Talipes*); Diabetes Insipidus (*Diagnosis*); Diaphragm (*Paralysis, Hysterical*); Eclampsia (*Diagnosis*); Fifth Nerve, Affections of (*Affections of Taste, Hysteria*); Hemiplegia (*Forms, Hysterical*); Hiccough; Hip-Joint, Diseases of; Hypnotism (*Therapeutic Uses of*); Insanity, Nature and Symptoms (*Etiological Varieties*); Joints, Diseases of (*Hysterical or Mimetic Affections*); Kidney, Surgical Affections of (*Symptomatology, Pain*); Micturition (*Frequency, Causes*); Mind, Education of (*Hysterical Children*); Myasthenia Gravis (*Diagnosis*); Paralysis (*Diagnosis*); Peritoneum, Acute Peritonitis (*Diagnosis*); Purpura (*Symptomatic*); Skin, Pigmentary Affections of (*Emotional Pigmentation, Hysteria*); Sleep, Normal and Morbid (*Morbid Dreams*); Spasm (*Clonic and Hysterical Spasm*); Spine, Surgical Affections of (*Railway Spine*); Spine, Surgical Affections of (*Caries, Diagnosis*); Spine, Surgical Affections of (*Hysterical Spine*); Syphilis (*Parasyphilis*); Syringomyelia (*Diagnosis*); Tabes Dorsalis (*Diagnosis*); Temperature (*Hyperthermia, Causes*); Tetanus (*Diagnosis*); Tetany (*Diagnosis*); Trades, Dangerous (*Lead Poisoning, Symptoms, Toxic Hysteria*); Trades, Dangerous (*Bisulphide of Carbon Poisoning, Symptoms resembling Hysteria*); Typhoid Fever (*Complications and Sequelæ, Nervous System, Hysteria*); Vision, Field of (*Hysterical Peripheral Limitation*).

Definition.—Hysteria, as its etymology indicates (ὑστέρα, uterus), was long considered an illness confined to the female sex; the ancients, indeed, regarded it as the result of a disturbance of the uterus seeking relief. Modern writers, however, have with one accord discredited the uterine theory of the malady. The term hysteria, nevertheless, though denied its original signification, is not on that account to be discarded, for, as Janet says, it has such a singularly interesting history that it would be difficult to replace it.

What is hysteria? A precise definition can scarcely be made, as it is not a disease in the ordinary sense, but rather a state of reaction and hypersensibility. If its manifestations are varied, it is no less true that they are all specially allied to affections of the nervous system. Briefly, hysteria is a neurosis of many and perplexing varieties, and of psychic origin, whose disorders often simulate organic disease. Many of the symptoms are variable and merely accidental, others are invariable and form the "stigmata of hysteria." For the sake of lucidity we shall group the symptoms according to the systems or organs of which they indicate functional derangement.

Etiology.—The appearance of a symptom of hysteria generally proves that the malady has already existed for some time, though latent. The name of an *exciting cause of hysteria* is given to any circumstance which suddenly reveals the malady, but the true cause of the disorder is an hereditary predisposition. If the real cause is single, the exciting causes are numberless. George Guinon, in his thesis, devotes several chapters to them. The *moral emotions*, fright, grief, anger, and other psychic disturbances, are the most frequent causes of occasional hysterical affections; and in every walk of life hysterical subjects are equally liable to attacks. *Traumatism* plays a very important rôle in its development; it acts in the way of a real suggestion, as much by the nervous shock as by purely physical causes,

creating a special nervous condition. To hysterical traumatism must be attributed the railway spine, the railway brain, and the traumatic neurasthenia of German authors. All accidents are apt to provoke manifestations of hysteria, as for instance bites, blows, earthquakes, surgical operations, etc. The severity of the traumatism is not so important as the mental impression produced. It is undoubted that *poisons*, both acute and chronic, are powerful agents; the commonest are lead and alcohol, sulphuret of carbon, tobacco, and morphia; these produce ordinary hysteria without peculiar characteristics. *Infection* also gives rise to hysteria, acting, according to Potain, like an internal traumatism. The commonest forms of such infections are typhoid fever, pneumonia, rheumatism, ague, blennorrhagia, and syphilis. Other general maladies, such as diabetes, sciatica, and gout, have the same exciting action. Certain morbid conditions favour the neurotic tendency, such as anæmia, and sexual excess and nervous disorders, such as disseminated sclerosis, tabes, and myopathy.

The true cause of hysteria is manifestly *hereditary predisposition*. Briquet's statistics prove that in nine cases out of ten hysterical parents have hysterical children. The cases may be traced to indirect as well as direct heredity; the subjects have had epileptic and neuropathic ancestors. A neurosis often passes over one generation. Grasset proves that scrofulitic, consumptive, arthritic, alchoholic, and syphilitic ancestors produce an hysterical race. Hysteria may appear at any age. With men and women alike it is most frequent between the ages of 19 and 25 years, frequently at the crisis of mental and physical puberty. It is rare before the age of 6 or 8 years, and exceptional after 40 years. The researches of Marie, Souques, and Chauffard prove that hysteria is commoner among men than among women in the lower ranks of society, probably because the artisan class is more exposed to traumatisms and poisons (alcoholic, etc.). Women suffer more in the upper and middle ranks. No race or climate is exempt; hysteria seems to have a marked predilection for the Jewish race.

Symptoms.—It will be convenient to discuss these in the following order: (*a*) convulsive attacks; (*b*) motor and sensory disorders; (*c*) visceral manifestations. It is well to bear in mind that in some cases numerous symptoms coexist, while in others the symptoms present themselves in an isolated manner—"monosymptomatic hysteria."

Hysterical Convulsions

Convulsive phenomena are frequent in hysteria, more especially in women. According to Pitres 82 per cent of cases in women have convulsions, and only 22 per cent in men. In men, however, the violence of the attack makes up for its rarity.

An attack of hysteria may appear in various degrees. A violent attack is called "hysteria major" or "attack de la Salpêtrière," a less violent attack "hysteria minor." The attack itself may present anomalies.

A. *Great Attack.*—Its different phases are shown in the following table borrowed from Charcot and modified by Grasset:—

Premonitory period	Prodromata Aura hysterica	Psychic trouble and hallucinations Disorders of the organic functions Disorders of mobility Sensory disorders
First period, or Epileptoid period	Phase at commencement Tonic phase Clonic phase Phase of muscular resolution	With movement With tetanic immobility
Second period, or Period of contortions and violent movements Clownism	Phase of contortions or disordered attitudes	
Third period	Period of emotional attitudes or plastic poses	
Fourth period	Period of prolongation. Period of delirium. (Delirium, hallucinations, zoopsia, disorders of movement.)	

Premonitory Period.—The symptoms consist of psychic phenomena, followed by somatic phenomena. Several days before the attack the patient is dull and depressed, and desires to be alone. In other instances there is increased nervous irritability, the patient laughs and cries, and is unreasonably agitated. He sleeps badly, and has dreams and nightmares; he has hallucinations attended with anæsthesia, he sees black and red animals, and his hearing is abnormally acute. His will-power at this stage is still strong enough to avert or arrest the attack. Other complications coexist with these psychic phenomena, the appetite is irregular and capricious, nausea and vomiting supervene, with hiccough and abdominal distension (tympanitis). The saliva is abundant, the quantity of urine increased, and the patient is troubled with flatulence, laryngeal spasms, etc. Palpitation, hurried breathing, alternations of flushing and pallor, sensations of heat and cold, also ensue. Other phenomena are cramps, shiverings, slight trembling of the legs, general weakness, and temporary contractures.

After this prodromal period the true aura makes its appearance, and marks the commencement of the actual attack. The ovarian aura is the most frequent, and merits a detailed description. The abdomen becomes painful at the level of the zone occupied by the ovaries; the pain, at first local, spreads over the whole surface of the abdomen, which is now tender to the touch; the disorder then rises to the throat, and the well-known sensation of the globus hystericus is produced, accompanied by palpitation and a sense of

tightness which forces the sufferer to tear off her clothing. The "hysteric fit" produces a sensation of suffocation and strangulation. The aura in certain cases begins in the epigastrium, instead of the ovarian zone, and follows the same course. A cephalic aura is marked by throbbing veins in the temples, singing in the ears, and specks floating before the eyes; such an attack often commences in the back portion of the brain (*clou hystérique*). In men the auræ belong to the testicular zones and the pseudo-ovarian zones.

First Period, or Epileptic Period.—This develops in three phases. In the first phase the aura is followed by shocks; without warning the patient loses consciousness, the breathing stops, the head rolls and falls back, the mouth is open, the face distorted, the eyes convulsed, the limbs twisted, the body heaving up and down. The patient now becomes rigid, the throat swells, the veins are swollen and projecting, the face is cyanosed and puffy, the features are rigid and a little froth escapes the lips. The arms are thrown up, the elbows out, the wrists bent, and the fists clenched; the lower limbs are extended, the knees pressed one against the other, and the feet pointed. The body is usually in a state of opisthotonos, more rarely in a state of emprosthotonos, or sideways in pleurothotonos. This phase of tonic contracture is usually brief, lasting only one or two minutes. Two phenomena are characteristic of it, namely, loss of consciousness and spasms in breathing. This tonic phase is succeeded by the clonic phase, as in epilepsy. The breathing recommences, irregularly and in hurried jerks; the whole body is shaken by rapid and extensive convulsive motions usually more pronounced on one side, the head oscillates, and the abdomen is distended. After a time the convulsions cease, and a phase of muscular resolution begins; this is the third period of the epileptic period. The patient is stretched on his back, his face contracted and the head sunk, the eyelids rising or falling from time to time, and the breathing regular and stertorous. The patient is now in a state of coma, interrupted by slight shocks and a degree of contracture; this phase lasts from two to five minutes.

Second Period, or Period of Convulsions.—Convulsions appear in two forms: (1) Contortions or clownish attitudes. (2) Emotional attitudes. The phase of contortions is characterised by disordered movements, attempts at wrestling, or trials of strength (clownism). The attitude called the *arc de cercle* appears during this phase, the body raising itself from the bed, and only resting on the head and the heels. The *arc de cercle* is generally anterior, more rarely posterior or lateral; the face is convulsed and grimacing. The movements of the body are more pronounced than in the preceding period, they simulate bowing, etc., and the limbs are distorted. The patient cries and shouts, tries to bite, and becomes so violent that he has to be held down by force. His attitudes correspond to his various emotions. There exists a transition from this phase to the following one, *the phase of emotional attitudes.* The patient expresses by attitudes his mental condition: anger, prayer, etc. Reminiscences of former hallucinations or scenes which occurred before his seizure, such as violin-playing, disputes, etc., powerfully affect his attitudes. When the attack reaches its height a kind of somnambulistic trance occurs, lasting from five to fifteen minutes. As the patient recovers he can often explain his attitudes, for he often remembers the hallucinations.

Period of Delirium.—In this last phase the patient suffers from delirium and hallucinations, greatly aggravated by outside influences, to which he was formerly indifferent. During this later phase of paroxysmal disturbance, hallucinations (zoopsia), paralysis, partial contractures, and epileptic shocks may supervene. Its duration is very uncertain, lasting hours and frequently days. The end of the attack brings with it a feeling of relief, and the patient resumes his normal condition. Accidents are rare, though cases of death have been known, occasioned by respiratory spasms. If the attacks are frequently repeated the condition is more serious, as the case then becomes one of true chronic hysteria (*état de mal hystérie*).

Such is an attack of *hysteria major* as described by the school of La Salpêtrière. Some writers deny its existence, and argue that it is merely a type of the disease artificially engendered by the surroundings of hospital life. This opinion is not tenable; the reality of this form of hysteria is only too true, and a consideration of its symptoms furnishes a key to the other more or less developed forms of hysteria which are constantly met with.

B. *Hysteria minor.* — This is the most frequent form of hysteria, and it also has three periods. During the pre-convulsive phase the prodromal phenomena differ very slightly from those of hysteria major; the ovarian aura, however, is wanting. The initial spasm seizes the hollow of the epigastrium; and the attack may often be arrested at this crisis. The period of convulsions, after a comparatively short tonic phase, during which one observes the swelling of the neck, the contortion of the face, and suspension of breathing, is followed by more rapid movements, hurried breathing, clenched jaws, and cries of anguish. This period of agitation soon yields to one of emotional attitudes, the attacks generally resembling one another. The crisis terminates in the phases of resolution and delirium, during which the patient neither speaks nor moves, and in which fits of crying, singing, or laughing may alter-

nate. After some little repose the patient can resume his ordinary occupations.

Varieties of Attacks. — In many cases the attack is not always typical; it is often modified either by the predominance of one period which obliterates the others, or by the accession of foreign elements. To illustrate, the aura may supervene without loss of consciousness, and may be marked by a crisis of palpitation or of suffocation, or sometimes by hydrophobic phenomena. The epileptoid attack sometimes simulates epilepsy, more especially as to frequency of attacks; or again it resembles Jacksonian epilepsy. Certain subjects suffer from vertigo like epileptics, and in some cases the vertigo simulates Ménière's vertigo.

The phenomena may be limited to those characteristic of the second period, namely, to the *convulsive* stage, and it is to this form of attack that maniacal excesses belong. In the third period the emotional attitudes always present the form of ecstatic emotion. In the fourth period the terminal delirium may present itself under various guises, to which the name of *folies hystériques* has been given. These last appear in somnambulistic form, and are always identical in the same patient. Lastly, the subjects may feel themselves impelled to exhibit the phenomena of *ambulatory automatism.* We must also refer to the *fits of sleep* which simulate coma, but are distinguished by the rigidity of the limbs and by trismus; they are attended by difficulty in breathing, which may be quickened or suspended, or assume the Cheyne-Stokes rhythm. During this sleep the patient hears sounds, but is incapable of response. This hysterical sleep may be compared to attacks of pseudo-syncope and hysterical fainting fits, and to the irresistible tendency to sleep which is so difficult to distinguish from narcolepsy. In several cases the attack resembles an apoplectic fit (*apoplexie hystérie*), and is accompanied by a hemiplegia either fleeting or permanent.

Sensory Disorders

Sensory disorders constitute one of the invariable and most conclusive symptoms in the diagnosis of the neurosis; they affect equally the mucous membrane, the skin, and special senses; sometimes by the diminution or abolition of sensibility, sometimes by hyperexcitability or hyperæsthesia.

A. *Anæsthesia.*—Anæsthesia has been long known. Witch-hunters and magistrates in the Middle Ages searched with care for "diabolic stigmata" in persons accused of sorcery, but it is only in modern times that the true significance of stigmata has been proved. The anæsthesia shows itself in 60 per cent of cases, but though a common symptom it is variable, and is sometimes complete, sometimes incomplete. Pitres has shown by the following table its different degrees:—

1. Total, bearing on all the sensory perceptions.	(*a*) Complete anæsthesia, properly so called. (*b*) Incomplete hypoæsthesia.
2. Partial, bearing only on certain sensory perceptions, the others not being affected.	(*a*) Loss of the sensations of pain, with the preservation of the tactile sensations: *Analgesia.* (*b*) Loss of the sensations of movement, preserving the tactile and painful sensations: *Thermoanæsthesia.* (*c*) Loss of the tactile and painful sensations, preserving those of movement: *Anæsthesia with Thermoanæsthesia.* (*d*) The isolated loss of electric sensations: *Electroanæsthesia.* (*e*) The preserving alone of the electric sensations: *Anæsthesia with Electroanæsthesia.*

From a topographical point of view anæsthesia assumes different forms. In exceptional cases it may spread over the whole surface of the body; generally it is partial and confined to one side of the body, and is then hemianæsthesia, which has a marked predilection for the left side, being three times more frequent on the left than on the right side; sometimes it appears in islets, insular anæsthesia; and lastly, it may be segmentary. It may be superimposed on paralysis or contracture, affecting a limb or portions of a limb. Like other hysterical phenomena anæsthesia is variable, and may be spontaneously modified either by some cutaneous irritant, such as electric currents, blistering, etc., or in a convulsive crisis by a suggestion or effort to pay attention. Hysterical anæsthesia in contrast to organic anæsthesia is compatible with the normal exercise of most of the reflex actions. The patellar reflexes, cremasteric and abdominal of Rossbach, are generally preserved, so also is the pupillary reflex action. Pricks practised on the level of the anæsthetised region are not attended with a flow of blood, but an appearance of a red aureola sets in, like nettle-rash, followed by a flow of limpid serum. Hysterical anæsthesia has another distinctive characteristic; it is attended with no conscious pain to the patient. The results obtained by an exhaustive diagnosis frequently differ even in the same subject; hysterical patients comport themselves like malingerers.

Anæsthesia of mucous membranes is frequent, being exhibited mainly in the mouth and pharynx, and to a less extent in the genital and anal mucous surfaces.

Ocular Anæsthesia is the commonest form of sensory anæsthesia. It may result in the sudden total loss of vision in one or both eyes, but this is rare; more frequently it causes no subjective visual disorder, but there is sometimes a central scotoma or even temporary hemiopia. On objective examination there is always found a restriction of the field of vision; this is concentric in character, in some cases symmetrical,

in others unequal, one or other side being specially affected. While the restriction of the field of vision is permanent, its limits vary from one moment to another during the attack, due to emotion or fatigue, *e.g.* under prolonged examination. There is no lesion at the back of the eye on ophthalmic examination. Besides limitation of the field of vision there is partial colour-blindness, chromatopsia. In pictures the patient can distinguish the perspective, trees, etc., but cannot see the colours; everything seems grey. Dyschromatopsia is very common; the loss of the colour-fields is elective, and is generally in a given order, the colours usually disappearing in the following order: violet, green, blue, yellow, and last of all red. The colour-fields of vision are inverted. Whereas in the normal condition blue has the widest field, in the abnormal condition red is the greatest. More rarely the disappearance of the colour-fields is in a different order, viz. violet, green, red, yellow, blue, and this modification of the colour-fields is a very important symptom in hysteria. Although dyschromatopsia is met with in other maladies, the order of colour disappearance is not the same. Thus in alcohol poisoning the red and green go first, and in tabes the red first. In the diagnosis of dyschromatopsia these facts have considerable value. Monocular dyschromatopsia presents one interesting feature: the patient affected with monocular dyschromatopsia for one colour sees this colour correctly by the use of binocular vision, a fact proved by the researches of Parinaud and Pitres. The limitation of the field of vision is accompanied by marked symptoms of asthenopia; if any object is looked at steadily the eyeballs burn and throb, and are wearied.

Other important symptoms appear side by side with these ocular disorders, *e.g.* diplopia or monocular polyopia, megalopsia and micropsia. Monocular polyopia is discovered as follows: a pencil is placed vertically near the patient's eye (the other eye being closed), and then is slowly removed ten or twenty centimetres; the patient perceives a second object on the temporal side as the pencil is moved farther off; later the two objects may recede and a third appear. This polyopia is largely due to a defect in accommodation. These ocular phenomena always accompany anæsthesia of the conjunctival mucous membrane and more rarely anæsthesia of the cornea.

There is *anæsthesia of taste* when the gustatory sensations are deranged; the sense of taste may be limited, perverted, or abolished. As an example of perversions salt may be mistaken for sugar, etc.

Anosmia is rarer than *ageusia*, and may be absolute or partial, or elective, where certain odours are perceived and others unperceived. The general sensibility of the lingual and nasal mucous membrane is sometimes increased and sometimes diminished. *Disorders of the auditory nerves* are sometimes subjective, sometimes objective. In the former, whistlings and singing in the ear may be present, and in the latter total or partial deafness. Rinne's test gives a positive result, and Schwalach demonstrated that the perception of sound lasts longer than when the patient is in his normal state. There are also variations of hearing; a patient may hear a whisper at some distance off, and he cannot hear a watch close to his ear. This perverted acuteness of hearing may be a very pronounced feature. Disorders of the aural organs, in common with those of the ocular organs, are irregular and variable. They often coincide with anæsthesia of the auditory nerve and tympanic membrane, but this coincidence is by no means absolute.

Anæsthesia attacks not only the superficial surface of skin and mucous membrane, but descends to the muscles, tendons, periosteum, and articular ligaments. The anæsthesia in the latter is evidenced by the fact that an unnatural amount of movement can take place without inconvenience to the patient.

The muscular sense is sometimes intact, sometimes altered; the patient loses the sense of the appreciation of weights, does not feel fatigue or the strain following an effort, and is insensible of the position of his limbs. These muscular troubles betoken a serious alteration in the sensory condition, and indicate some degree of anæsthesia.

B. *Hyperæsthesia.*—This is rarer than anæsthesia, and as a rule is more difficult to recognise; it differs also inasmuch as it causes much suffering to the patient. It may be general or confined to one limb, its area being limited to one or two centimetres square. These zones, called *hysterogenic points or zones*, may be situated in the abdomen, about the ovarian or epigastric region, the groin, the thorax, the breasts, the clavicular region, the spinal column, from the top of the throat to the eyeballs, and the malar region. These zones may be bilateral or unilateral, and they are usually first observed near the anæsthetic areas. An attack of hysteria may be brought on by rubbing or pressing these zones, in which case the zones are called spasmogenic; the same means also may be used to relieve the attack, in which case the zones are called *spasmodic* or spasmofrematic. The same zone can be alternately spasmogenic when it is gently pressed, or spasmofrematic when it is vigorously rubbed. As a general rule the zones situated at the level of the thorax are spasmogenic, and the epigastric and ovarian zones are spasmofrematic. These zones are limited, the bounds of hyperæsthesia are clearly defined, and the zones are liable to appear and disappear spontaneously; they can be momentarily suppressed by distracting the patient's attention, or by suggestion, or the influence of electrisation,

particularly of static electricity. At the commencement of the attack these hysterogenic zones are painful, and there is a feeling of constriction and bruising. They may be superficial or deep-seated, and are cutaneous in the sense that gentle friction will excite an attack. These hyperæsthetic areas are also sometimes found over the following glandular organs, the mammary gland, the testicles, and the ovaries; the ovarian zone is the most frequent. The seat of the hyperæsthesia is generally the gland itself, for certain authors have traced out the displacements of this zone as the ovary ascends during pregnancy. Hyperæsthesia, though less constant than anæsthesia, is an important factor in the induction of the attacks, and it is more often the cause of violent convulsive attacks.

Up to the present we have only taken notice of the objective disorders of hyperæsthesia, so to speak, now let us consider the subjective—the different *algies* or hyperalgesias of Pitres. These include a variety of phenomena, and give rise to many painful symptoms. Heptalgia, enteralgia, pleuralgia, ovaralgia, and testicular irritability may be prominent features; ophthalmic megrim is more uncommon, and can be distinguished from the organic form by the absence of hemiopia. Other neuralgic affections may occur, such as sciatica. No matter where the seat of pain may be, hysterical neuralgia can easily be distinguished from ordinary neuralgia. The fits of pain do not come on spontaneously, but are provoked by accidental excitement, etc., and may reach an extraordinary intensity if the descriptions of the patients can be believed.

Cephalalgic hysteria, real or superficial, may be limited to one region (*clou hystérique*). Certain patients suffer intense pain, which returns periodically in the evening or early morning hours, and is sometimes mistaken for syphilitic cerebritis or tumour on the brain. It is accompanied with giddiness, sickness, sleeplessness, etc., which may simulate meningitis (meningitis, pseudo-hysterical). Spinal hyperæsthesia may be accompanied with contracture, and simulate a *mal de pott hystérique*; if it follows a traumatism this symptom may give the idea of a fracture of the spine. Other hysterical manifestations have been mistaken for arthritis, and have induced mistaken surgical intervention. Charcot maintains that arthritis of the knee is the most frequent. In M. Focas's cases the arthralgia attacked the knee thirty-eight times, the hip eighteen times, the wrist eight times, the shoulder four times, and the instep twice. These arthralgias are always accompanied by contractures at the elbow and knee, where the position assumed is one of flexion; at the shoulder there is adduction, which causes a twisting of the trunk. Hysterical coxalgia is one of the most interesting of these arthralgias, as it may closely simulate true coxalgia, and as in the latter, the patient is unable to walk, and the least movement of the articulations causes pain. The limbs may be in a state of flexion or abduction. It requires a close examination to distinguish true coxalgia from hysterical coxalgia. In the latter, percussion of the great trochanter causes no pain in the knee-joint. Further, the sensitiveness of the skin in the corresponding region is modified; the zone of pain assumes the shape of a triangle, whose apex is situated over the end of the sacrum, and extends to the folds of the groin and buttock. Hysterical coxalgia develops more rapidly than ordinary coxalgia, and an examination under chloroform shows no subluxation, indicating an organic lesion; in cases where amputation has been resorted to, owing to an erroneous diagnosis, the articulations have been found to be quite sound.

C. *Dysæsthesia.* — Having spoken of hyperæsthesia and anæsthesia, a few words may be said of the paræsthesiæ. *Haphalgia* is the name given by Pitres to the sensory condition, in which a feeling of intense pain is created by contact with certain substances (such as copper, brass, etc.), which in a normal state only produce the sensation of touch. *Allochiria* is the name Obersteiner gives to the condition in which the patient feels on one side of the body the impressions received on the other side.

Motor Disorders

These disorders may be permanent or temporary; permanent disorders must be carefully sought for, as they often frustrate efforts to find them. To illustrate, the hysterical patient may have slight paralysis which scarcely affects him, and is only perceptible by the use of the dynamometer. This muscular inertia induces disorders which the patient is unable to account for satisfactorily. For example, his legs give way under him when walking, just as they do in tabes.

Amyosthenia may be local or general, and affects the muscles more frequently on the right side than on the left, but varies from one moment to another, being influenced by the attack, by emotion, and by the æsthesiogenic agents. There is no pronounced modification of the tendon reflexes; they are generally normal, though sometimes weakened and sometimes exaggerated. The muscular feebleness has one striking peculiarity, it is only apparent in voluntary and not in automatic motion. In certain cases the amyosthenia has the appearance of akinesia algera. Another well-known hysterical phenomenon (Lasegue's symptom) must be studied along with amyosthenia, the patient cannot move unless he looks to see what he is doing; for example, C. Bell tells of a woman who could not hold her baby in her arms unless she looked at it. This phenomenon is accompanied by a tactile and muscular anæsthesia of the weakened limbs; in other instances the patient cannot move at night in the dark. Individuals suffering from

total anæsthesia cannot stand upright if their eyes are shut; no movement can be made unless the patient is looking at the anæsthetised limb, otherwise it remains in its former position (partial catalepsy). Pitres puts this fact lucidly before us; a movement is begun with the eyes open, and is arrested if the aid of vision is withdrawn. Other disorders of movement may be noted; *syncinæsia*, if the patient is told to lift *one* arm he lifts both; *allocinesia*, the patient raises the opposite arm from the one he is told to lift; *heterocinesia*, where the movement executed is the contrary one to the movement he is shown.

Paralysis and Contractures.—Paralysis, contractures, spasms, etc., are the temporary motor disorders of hysteria. They affect both the organic and voluntary muscles; disorders of the organic viscera will be studied in connection with the affection of the viscera, and only voluntary motor disorders will be here described.

Paralysis and contractures proceed from the same causes, and occur either at the end or during an attack. Very occasionally they precede the attack, in which case they are transient, but if they accompany the convulsive attack they may be much more obstinate. At times the predisposing cause is some local inflammation (congestion, laryngitis, etc.). The attacks affect the moral emotions most powerfully. Traumatism is influential in producing paralysis (*hystero-traumatism*); it need not necessarily be very violent, and there often exists very little relation between the intensity of the trauma and the degree of the motor trouble. The development of the paralysis is preceded by an interval of "meditation," during which the sufferer dreams incessantly of the accident of which he has been the victim; at the end of a time, varying from fifteen days to three months, paralysis or contracture appears; the force of the mental shock is thus seen to be greater than the physical, hence the term psychic paralysis, or paralysis of the imagination (*bei einbeildung*).

Hysterical paralysis develops sometimes slowly, sometimes rapidly, sometimes like a blow or ictus. Wherever it appears it has a certain number of ordinary characteristics. (1) It is usually partial, the limbs still retaining a certain power. These paralytic strokes are not always attended by amyotrophies, nor by vasomotor troubles and œdema. (2) The temperature is slightly lowered about the affected part, the electric reactions are scarcely modified, and the tendon reflexes are normal. (3) Another typical characteristic of this paralysis is the appearance of a segmentary, anæsthetic zone. (4) The muscular sense is numbed. Hysterical paralysis presents many variations, some astonishing in their intensity and duration, and lasting sometimes hours, sometimes years. The termination takes place on recovery, and may be sudden or gradual, or may give place to a contracture. In some cases the paralysis is systematic, and only affects certain movements. Hysterical paralysis is distinguished from cerebral paralysis by its irregularity and the intensity of the sensory disorders; and from organic paralysis by the absence of any considerable muscular atrophy or disorders of the reflexes. The locale of paralysis is varied; with a monoplegia the paralysis is flaccid, and the limb feels inert. The *sensory disorders* are the following: in the upper extremity the anæsthesia is *en gigot*, with a slight encroachment on the thorax before and behind; in the lower extremity the superior limit of the insensibility is marked by a line passing through the fold of the groin, the iliac crest, and the insertion of the gluteus maximus, leaving out the genital organs and the sacrum. Hemiplegia is the ordinary form of hysterical paralysis, which affects most frequently the left side, and is accompanied by the characteristic gait described by Todd: the lower limb is inert and drags like a useless member, and, unlike organic hemiplegia, there is no power of motion or circumduction, and the feet drag on the ground; hemianæsthesia is always observed. The face generally escapes, though occasionally it is affected in the course of hysterical hemiplegia. In some rare cases this reproduces the *Gubler Millard symptom.*

The existence of hysterical facial paralysis was long doubted, but is now an incontestable fact. It may be accompanied by *glossolabial hemispasm* (*vide* p. 315). It is seldom very acute, being more frequently a paresis rather than true paralysis; it has one outstanding characteristic, viz. a remarkable disconnection in the motor faculties, the patient being able to laugh or cry, but possessing no power of voluntary action. *Hysterical paraplegia* is sometimes observed. It may be associated with a flaccid state of the muscles, or with rigidity, and in the latter case may closely simulate serious organic disease of the spine. It is distinguished from organic paraplegia by the absence of subjective pains and by the particular type of anæsthesia. In certain cases paraplegia simulates tabes, "hysterical pseudo-tabes"; the association of tabes with hysteria is not otherwise an exception, the two maladies often appearing separately on their own account (hystero-tabes).

The occurrence of paralysis of the ocular muscles, although admitted by certain writers, is problematical.

Contractures develop suddenly or gradually like paralysis, and consist in motor weakness and persistent stiffening of the muscles. The contracture involves the antagonistic muscles, and only relaxes under the influence of chloroform. A segmentary anæsthetic zone is present as in paralysis, the reflexes are rarely augmented, and epileptic tumours are rare, though there is hysterical trembling. A common form of con-

tracture is one which is slight and disappears during sleep. Another type is accompanied by considerable hyperæsthesia with spontaneous paroxysms and pain, together with painful anæsthesia and serious disturbance of the general sensibility. Contractures have a tendency to remain; if they improve they may reappear suddenly on the slightest provocation. With the neuro-muscular system in a state of hyper-sensibility, a very slight moral or physical shock may set up a contracture. In this class of patient the contracture seems to be waiting to be induced; this peculiarity is called *diathesis of contracture.* This contracture easily reveals itself by percussion of the tendons, by massage of the muscles, by faradisation and compression with Esmarch's bandage. *Diathesis of contracture* is not confined to hysteria, but is met with in cases of organic degeneration of the pyramidal tract. Hysterical contractures must not be confounded with simulated contractures; these latter only occur after a great effort, and betray themselves by the acceleration of the respiratory movements. In organic contractures the sensory disorders are slight, the tendon reflexes are always exaggerated, and the fibrillary twitchings never cease. Hysterical contracture even in its external aspect differs materially from one of organic origin; in this latter the rigidity is slight and may be overcome within certain limits, whereas in hysterical contracture the rigidity is such that it cannot be overcome; the attitude of the limb is also more exaggerated, and the flexion more pronounced than in organic contracture. Contractures may assume different types. As in paralysis, hemiplegic and paraplegic, we shall only mark monoplegic and limited contractures; hemiplegic and paraplegic types are always accompanied by a slight degree of paralysis. When the contracture involves an upper limb its aspect is characteristic, the arm is adducted, the elbow bent, the hand may be closed or opened, and the fingers may be placed as if they held a pen. At other times the wrist is strongly bent and the fist closed. In monoplegia of a lower limb the appearance is one of extension; the contracture is often localised to the foot—the hysterical club-foot, in which the toes are sometimes stretched out, sometimes curled in. Contracture of the ankle is rare. Among the contractures confined to a muscular group we must mention the very rare trismus, the glossolabic hemispasm, the contracture of the muscles of the eyes, blepharospasm, and wry-neck, or torticollis.

Glossolabial hemispasm, already referred to, is frequently conjoined with paralysis and gives the sufferer a very singular expression. In repose the mouth is twisted, the nasolabial furrow deepened, the eyebrows lowered, and slight muscular tremors agitate the face from time to time. If the patient is ordered to open his mouth, or to laugh, or put out his tongue, the spasm becomes intense, the nasolabial furrow is curled up, the teeth are uncovered more on the contracted side than on the sound side, the tongue is thrust out with some inclination to the contracted side, or may be pushed against the cheek inside instead of pushed out. The spasm may extend to the muscles of the eyelids and of the neck, and in exceptional cases it is bilateral.

Blepharospasm may exist alone or in conjunction with the glossolabial hemispasm. There are various forms. In the clonic form there is a constant trembling of the half-closed eyelid. In the permanent or tonic form the eyelid is folded and is difficult to close or raise; if the eyeball is examined it is seen to roll from side to side. In the pseudo-paralytic form there is slight ptosis, without wrinkling of the eyelid or brow, and from time to time there are slight convulsive quivers. This false ptosis is distinguished from paralysis of the levator palpebræ superioris by the lowering of the eyebrow on the side where the spasm exists; in true paralysis it is lowered and accompanied with anæsthesia of the conjunctiva and of the eyelid. *Contractures of the eye-muscles*, though rare, do exist; the disorders of accommodation are attributed to contracture of the Brucke muscle, such disorders as monocular polyopia, micropsia, and megalopsia. In convergent and divergent strabismus the contracture of the motor ocular muscles may simulate ophthalmoplegia.

Among other local contractures *torticollis* may be mentioned; in this contracture the twisting of the neck cannot be prevented, a fact which distinguishes it from paralytic torticollis, a much rarer disease. Torticollis, when associated with subjective pains of the mucous membrane, may be mistaken for cervical caries. Contractures of the muscles of the trunk give rise to scoliosis and kyphosis.

The motor affections which we have still to investigate are chorea, tremblings, saltatory spasms, and astasia-abasia.

Hysterical chorea belongs to two classes: one very rare, arrhythmic chorea, in which the gestures and movements are disorderly, as in the chorea of Sydenham; the other more frequent, rhythmic chorea, in which the shocks attack the limbs, the face, the neck, or the half of the body (hemichorea). It may be thus described: a series of regular cadenced shocks, followed by paroxysmal fits, lasting generally from fifteen to twenty minutes, rarely one or several days. These rhythmical co-ordinated spasms reproduce gestures associated with some action, such as dancing (*chorea saltatoria*), swimming (*chorea natatoria*), a smith beating his anvil (*chorea malleatoria*). (*See* article "Chorea.")

The hysterical tremblings following an attack or fit are generalised or limited, and are characterised by an intense polymorphism, causing them to resemble all the recognised

varieties of tremors. Dutil has described several leading types: 1st, A type of rapid oscillations (eight to ten shocks per second), slight but continuous, suggesting alcoholic tremors, and the movements in Basedow's disease (exophthalmic goitre). Sometimes these tremors are associated with other symptoms of exophthalmic goitre, and the two diseases may simulate each other. 2nd, A type with a mean rhythm ($5\frac{1}{2}$ to $7\frac{1}{2}$ shocks per second), allied to mercurial quiverings and those of disseminated cerebro-spinal sclerosis; this trembling when it attacks the lower limbs simulates epileptoid tremors. 3rd, Slow tremors with severer shocks (4 to $5\frac{1}{2}$ shocks per second), resembling the trembling of paralysis agitans.

The saltatory spasm was closely studied by Brissaud, and consists in a series of successive contractures of the extremities with bending of the legs and thighs, following some excitement, slight shock, or agitation. The patient leaps in an odd way when he walks, as if he trod on hot cinders; the muscular force is intact. This spasm is generally bilateral; if the patient is in bed, a slight irritation, percussion of the tendons, or twisting of the foot over the leg, may provoke this spasm. This dancing spasm may be compared to the *paramyoclonus multiplex* and *electric chorea*, as both are in the list of hysterical accidents.

Astasia-abasia may be defined as the suppression or disorganisation of the co-ordinated movements concerned in walking or standing upright. As may be inferred from this definition, it usually affects the lower limbs, and is rare in the upper limbs, where it betrays its existence in difficulties in writing or drawing. The features are very peculiar. The patient can leap, run, or hop, though he cannot walk or stand upright. Abasia may exist alone. There are two forms of astasia-abasia: the one paralytic, in which walking and standing upright is impossible, for the legs give way under the patient; the other ataxic, in which the symptoms only appear when the patient wishes to walk and stand upright. Astasia-abasia has a tendency to return.

Disorders of the Respiratory Organs

Dyspnœa is frequent in hysteria, and manifests itself under the influence of emotion or without any apparent cause. Its aspect is variable, it is sometimes due to a spasm of the glottis, sometimes to difficulty of breathing arising from paralysis or contracture of the diaphragm, more frequently to *tachypnœa* (from 170 to 180 respirations per minute) without uneasiness or acceleration of the pulse. The *hysterical cough* may accompany dyspnœa, or may be an isolated symptom. In not a few instances these symptoms have led to an erroneous diagnosis of tuberculosis. This cough has marked peculiarities; it is rasping, resonant, without expectoration, rarely permanent, and habitually paroxysmal. The fits may be isolated or grouped, and follow always the same rhythm, from 3 to 4 shocks; it is rhythmical, a true equivalent of the hysterical attack, and accompanied by similar phenomena. It is tenacious and fierce, and has special variations of sound, often resembling a dog barking, a wolf howling, a hen cackling, or a dove cooing. These phenomena of laryngeal spasm occur in epidemic form in convents, schools, etc. With these spasms of expiration there may be spasms of inspiration, fits of yawning, sneezing, and uncontrollable laughter.

The disorders of phonation are many and various. In the first rank comes *hysterical aphonia*, which develops suddenly, and is a veritable pure motor aphasia, without mental impairment; it may sometimes be accompanied by deafness and even agraphia. This disorder has a central origin, and is unconnected with any lesion of the muscles of the tongue and lips. It may last a long time, even years. The hysterical stammering which precedes or follows the aphonia results from veritable polysyllabic aphasia, and can, in many instances, be traced to spasms or contractures.

In *aphonia* the patient can only speak in a low voice or whisper, but articulates well. It often happens, when loud speaking is impossible, that the cough is sonorous and singing is possible, and it should be noted these patients can talk in their sleep. There is contracture or paralysis of the vocal cords. *Phonophobia* is less common, and consists in a fear of speaking too loud. In this condition the patient can only whisper, and any louder speech brings on an attack of pain in the larynx.

Disorders of the Digestive Organs

Gastric hysteria is important, and is frequently the forerunner of other visceral manifestations. *Dysphagia* is the most frequent phenomenon, and is due to spasm of the pharynx and œsophagus. Contracture of the œsophagus betrays itself by the food being returned immediately after ingestion. The initial stage of dysphagia is curious, as certain solid foods pass easily, although liquids are rejected. Bouveret describes a strange case, in which the patient had clonic spasms of the muscles of the pharynx repeated 30 or 40 times a minute—a condition which he regarded as hysterical dysphagia.

Hysterical vomitings often result from contracture of the stomach. They begin immediately after the ingestion of food, and are more or less continuous, often painless, and followed by anorexia.

Hysterical gastralgia is caused by an hysterogenic zone in the stomach or gastric region. The reception of food causes violent pain, often in paroxysms, accompanied by palpitation,

dyspnœa, retching, and hiccough. This is a real gastric attack, sometimes so violent as to closely simulate a gastric crisis in locomotor ataxia. When there is gastric atony the sensory disorders are slight, food is vomited some time after ingestion, and the patient suffers from a feeling of repletion in the stomach. Hæmatemesis sometimes appears, and when accompanied by pain may be attributed to ulceration of the stomach. This symptom is not uncommon in hysteria, and is called *hœmorrhagie pituite hystérique.* Mathew and Milian have described the rejection from the mouth of a certain quantity of bloody fluid (generally in the morning), red and acid like currant juice, and composed of ten parts saliva to one part blood. The appetite is perverted, sometimes ravenous, more often anorexied. *Anorexia* is the name given to the systematic refusal of food, and in some cases it is a grave and most troublesome symptom. The patient refuses to eat either from caprice or disgust with life, or from other cause which the patient cannot define. Anorexia is usually total, but it may be partial and refer only to certain foods. It is sometimes variable, transitory, and slight, and at other times just the opposite, and is then accompanied by other symptoms, the chief of which is a slight degree of mental excitement. Later, a state of debility follows, the patient keeps his bed in a half comatose state, the muscles of the neck are paralysed, the extremities are flaccid and cold, he becomes emaciated to a skeleton. It has long been matter of belief that hysterical patients can live without eating, but this is erroneous. The state of the digestive secretion of hysterical patients is variable, the gastric juice being sometimes normal, sometimes weakened in its digestive action. (*See* article "Neurasthenia.")

The intestinal phenomena arc, as a rule, less noticeable than the gastric phenomena. Constipation is the rule, but somctimes attacks of paroxysmal diarrhœa coincide with the vomiting. A mucous enteritis may be met with in these patients; eructations and flatulence may be perpetual, and almost rhythmical. Tympanites is specially important from its frequent occurrence and its often mistaken diagnosis. When pronounced, the abdomen is swollen, painful on palpation, and percussion gives a tympanitic sound, the breathing is laboured, food is vomited, and the patient may present the appearance of tuberculous peritonitis. In the paroxysmal form the aspect is that of acute peritonitis, the visage is drawn, the vomitings are persistent, constipation is obstinate, and there is a good deal of fever; this is *hysterical pseudo-peritonitis.* At other times the hysterical phenomena simulate fæcal vomiting. Tympanites may be local, and give rise to small tumours, and is not infrequently the cause of an erroneous diagnosis of ovarian, gastric, intestinal, or hepatic tumours. Tympanites also gives rise to pseudocyesis, specially in those women who crave for children, or those who dread having them. An examination under chloroform alone can solve the question.

Intestinal hysteria occasionally takes the form of appendicitis, the *spurious appendicitis* of Brissaud; rectal spasms may simulate stricture of the rectum. Before passing from the digestive disorders we must mention false hepatic colic of hysterical origin. By some writers jaundice is said to be caused by a similar spasm of the biliary passages.

Generative Organs and Organs of Secretion

Disorders of the Urinary System.—As we shall later study the disorders of secretion, we shall now confine our attention to the phenomena connected with the organs of excretion. Spasm of the urethra is a not infrequent symptom of hysteria, and when attended by slight hæmaturia may be erroneously diagnosed as renal colic. The bladder may be the point from which painful phenomena arise (cystalgia). Incontinence of urine is uncommon; retention of urine is much more frequent, caused sometimes by spasms or sometimes by paralytic anæsthesia of the mucous membrane of the bladder. To test what form of retention exists, catheterism must be resorted to. If the bladder empties slowly, the water only coming drop by drop, paralysis is indicated; while in spasm of the neck, when the obstacle is passed there is a rush of urine. This spasm of the neck of the bladder may be mistaken for stricture of the urethra.

Disorders of the Genital Organs.—In men hysteria is rarely to blame for genital disorders, except testicular anæsthesia. Many writers have shown the possibility of an hysterical epididymitis. In the case of women the severe neuralgic pains of the pelvis described by various physicians ought to be ascribed to neurotic ovaritis. Disorders of menstruation, amenorrhœa, dysmenorrhœa, and membranous metrorrhagia are frequent, and hysterical attacks have an influence on these disorders. Vaginismus may exist alone or conjoined with a rectal spasm; sexual excess is not such a prominent feature as is often supposed. Indeed it is not uncommon in cases of anæsthesia of the mucous membrane to find hysterical patients quite indifferent and insensible to sexual relations.

Disorders of Secretion.—Among these disorders may be named *salivation*, *galactorrhœa*, *hyperidrosis*, general and local, and lastly, *polyuria*, which merits a special study. The polyuria which follows a convulsive crisis has long been observed. It has been suggested that permanent polyuria of an hysterical nature can be induced by hypnotism, and proofs of this abound. The quantity of urine passed is considerable, at least ten to nineteen litres, and as much as twenty-nine litres being passed daily; polyphagia and

polydipsia are its usual attendants, and give the impression of diabetes. Polyuria is a serious and obstinate symptom, and is usually worst in men. At other times, instead of being exaggerated, the flow of urine is diminished, there is *ischuria*, sometimes simply *oliguria*, sometimes almost total *anuria*; even when the suspension of the flow of urine is complete no uræmic symptoms appear. Vicarious phenomena, such as vomitings, sweating, and diarrhœa, are not uncommon, the secretions usually containing an appreciable quantity of acid and carbonate of ammonia.

Disorders of Circulation and Trophic Disorders

Hysterical patients often complain of different cardiac troubles, palpitations, and *angina pectoris*; these differ from organic attacks of angina, in that they are the result of excitement and fatigue, and last some time. Sometimes pain with cutaneous irritation is the dominant phenomenon (neuralgic form), sometimes vaso-motor phenomena are the most prominent, the face becoming pale, thin, and livid (vaso-motor form). Such an attack may terminate with definite cardiac phenomena, notably increased pulse-rate with tendency to syncope; in other cases it terminates in a burst of tears or laughter. Other vaso-motor symptoms are common, more especially flushing and pallor and extreme sensitiveness to cold.

Dermographism, often met with in other nervous affections, is well known in hysteria, and is easily demonstrated. If we draw a line along the skin with the point of a pencil an artificial nettle-rash speedily develops. This takes the form of a broad band, the centre of which appears pale and œdematous, with a zone of congestion on either side. This may frequently be demonstrated after a convulsive attack. Certain hysterical subjects are in a state of vaso-motor diathesia analogous to diathesia of contracture. At a more advanced stage the neuro-vascular disorders express themselves by local *asphyxia of the extremities*, *erythromelalgia*, and lastly *hæmorrhagia*. Ecchymoses may develop under the skin, appearing equally on the ocular mucous membrane (tears of blood), nasal (epistaxis), auricular (otorrhagia), vesical (hæmaturia), uterine (metrorrhagia). To this list of vaso-motor disorders bloody sweats fall to be added.

The trophic troubles are no less interesting; among them are *bullous eruptions* of the pustular variety presenting sometimes the appearance of pemphigus, sometimes that of zonæ or herpes. These have been described as hysterical eczema and herpes. Pigmentary stains sometimes develop after some mental emotion or crisis. But the most important cutaneous trophic disorders in hysteria are œdema and gangrene.

Hysterical œdema is always unilateral and limited to one limb; it is very rarely general, simulating *anasarca*; it appears often in limbs already the seat of hysterical contraction. The skin affected by œdema is smooth and shining, the colour deep blue or violet, constituting the so-called blue œdema; the local temperature is lowered, the œdema is hard and does not yield to pressure. Blue œdema lasts a long time, generally several years. *Hysterical gangrenes* may be symmetrical and attack the extremities; they are most frequently in isolated plaques. Trophic disorders also attack the cutaneous appendages; it is noticed that the hair falls out, and the teeth and nails become friable.

The trophic troubles which attack the uterus deserve special attention. The *hysterical uterus* consists in a temporary or permanent enlargement of the organ, coming on suddenly and accompanied by considerable hyperæsthesia of the skin. The colour of the skin may be normal, or red, or even violet. In certain cases considerable œdema is present, and gives the feeling of a tumour. In many instances of hysterical swellings of uterus or breast the patient is operated on under the impression that there is a malignant growth; the analogy with cancer is made complete when there is a gangrenous plaque on the surface of the solid œdematous area. This form of trophic disorder, like œdema, may last a long time. Trophic disorders not only attack the skin and subcutaneous cellular tissues, but even involve the muscles; *muscular hysterical atrophy* is more or less considerable, and is not accompanied in the majority of cases with any modifications of idio-muscular excitability. The electric contractility is generally diminished, but the reactions of degeneration do not often appear in this form of muscular atrophy. This atrophy sometimes develops alone, but more frequently follows hysterical hemiplegia or monoplegia. The whole limb may be affected or only a portion, and it requires a longer time to recover than a case of simple hysterical paralysis. The tendons are also subject to trophic troubles, *fibro-tendinous retractions* may continue after the paralysis and contractures have disappeared; they are sometimes accompanied by the thickening of the soft tissues and necessitate surgical intervention.

Disorders of General Nutrition

General nutrition can be modified in hysteria in certain directions. It has long been believed that during the paroxysms of hysteria, nutrition is carried on under other than normal conditions. This is not the case: the character of the secretions proves that in hysteria the nutrition is normal. But there is some alteration after the attacks; an examination of the urine proves that nutritive changes then differ from those in a normal state. The alterations observed are commonly called the *urinary formula of hysteria*. If the urine collected in the twenty-

four hours after the attack be examined, there is found a reduction of the average amount of uric acid and phosphates, with inversion of the formula of these latter. The total solid, which normally averages from 40 to 52 grammes, is lowered to 35-38 grammes; the uric is lowered to 13 gr. instead of being 20 gr.; the phosphoric acid is 1·2 gr. instead of 2·2 gr., as in the inter-paroxysmal period. Whilst in normal urine the soluble phosphates and alkalies are as 1 to 3, after the attack they are as 1 to 1. This modification is called inversion of the phosphatic formula; the same urinary formula is found in attacks of partial or ordinary epilepsy, chorea, delirium, stupor, and other mental disorders.

The study of general nutrition leads us to observe *hysterical fever*. Its existence has been disputed, as a fever can be so easily simulated, but recent researches undoubtedly prove its existence. The variations of temperature are identical with the different known forms—remittent, intermittent, continuous, and sometimes polymorphic, changing quickly from intermittent to continuous. The rise of temperature may be considerable, reaching 41°, and even more. From a clinical point of view there are two very distinct forms: one in which hyperthermia exists alone, without other symptoms—the so-called hysterical fever—often accompanied with convulsive phenomena; the other form is accompanied by certain visceral disorders, and simulates other maladies, such as typhoid fever, meningitis, pulmonary tuberculosis, peritonitis, and malaria. This hysterical fever differs from infectious fevers in its irregularities, the absence of the usual serious symptoms, and its resistance to antipyretics; it should be considered as equivalent to an attack. The diagnosis of hysterical fever is a diagnosis by *exclusion*, but it should only be arrived at after careful consideration, as many organic disorders begin with hysterical manifestations.

Disorders of the Mental State

We have already spoken of the delirium which follows an hysterical attack, and we shall now study the mental condition between the paroxysms. This mental condition is one characterised by modifications of memory and will power. Derangements of memory are frequent, and cause the patient to make conflicting and contradictory assertions. Pierre Janet has made an exhaustive study of *amnesia* and its different modifications. (1) It is systematised, not confined to one given period in the life of the individual, but to a group of recollections; for example, it causes an individual to forget a foreign language or proper names. (2) It is localised, and causes the individual after some severe shock or accident to forget events accompanying or preceding the event. (3) It is generalised; the individual has then to be taught everything like a child. (4) It is anterio-retrograde, that is to say, the individual forgets events as soon as they happen. In spite of this amnesia the intellect remains intact, but the power of attention is enfeebled.

The will is often as much disturbed in hysteria as the memory; automatic actions are unconsciously accomplished, but voluntary actions, such as a new piece of work, are impossible. This loss of will power is very marked in hysteria, and explains the facility with which the patient responds to suggestion. The emotions and affections are often disturbed; sometimes the hysterical patient is excited, easily affected, and exceedingly irritable; at other times he is indifferent, sad, melancholy, and depressed; in both cases the emotional reaction is exaggerated and out of proportion to the cause. Hysterical subjects are sometimes accused of being liars, hypocrites, dipsomaniacs, etc., but these faults ought rather to be placed to the account of the mental degeneracy resulting from hysteria than ascribed to the subjects themselves. The question of legal responsibility in hysterical mental troubles is a difficult one. If criminal acts are committed during a paroxystic crisis the personal irresponsibility is incontestable, but if they take place during an interparoxystic period the personal irresponsibility is not quite so clear.

Clinical Forms of Hysteria.—Hysteria may appear in a convulsive form, or in a monosymptomatic form, in which case stigmata should be carefully searched for to prove the diagnosis. The clinical picture varies according to age; the convulsive reactions are less violent in the old—in whom they exist often in a masked state. The case of children is different, and merits a special description.

Infantile Hysteria.[1]—Infantile hysteria has very special manifestations owing to the brain conditions present in infancy. During infancy the brain develops very rapidly, the convulsive reactions are numerous, and at this period there is a continual growth of new and violent sensations; the nervous system is in a state of constant erythism. Education is an all-important factor in the nervous development of the child, and should the parents be of a changeable and unstable character, and the servants coarse and unintelligent, the child's predisposition to nervous disorders will be intensified. Such causes as abnormal physical growth, over-study, helminthiasis, and teething, all tend to develop these nervous disorders in the predisposed subject. The hysteria of early infancy must be distinguished from that of later years and adolescence. With the nursling many disorders attributed to teething are really hysterical. Not a few nervous disorders encountered in infancy where the disturbance exceeds in intensity the degree of reaction normal at that age, and which cannot be explained by an

[1] See also "Hysteria in Children," p. 325.

intoxication or infection, must be regarded as hysteria. If during infancy hysteria is rare, it becomes more common as the years go on, and clothes itself in very curious forms. "Who has not seen," says Grancher, "the little ones of four or five years playing with their dolls, and already showing in their shining eyes, their caressing and feline attitudes, their coquetry, and their malice, a neuropathic tendency only requiring opportunity to develop?" From the ages of eight to nineteen years, the disease may develop with most astounding symptoms, but is of shorter duration than in adults; as Charcot says, "Hysteria has no hold on a child." In grave cases infantile hysteria is monosymptomatic. The most frequently observed phenomena are vertigo, cephalalgia, stammering, fits of yawning, digestive disorders of a gastric nature, polyuria, and astasia-abasia. Trophic troubles such as œdema are not uncommon. Ocular disorders, save the narrowing of the field of vision, are seldom met with; hyperæsthesia governs to the exclusion of anæsthesia. Coxalgia, the malady of Pott, and hysterical torticollis, are habitual manifestations of this hypersensibility. In a less advanced degree hysteria expresses itself by cerebral irritation, dreaming, nightmares, and hallucinations; there are also digestive troubles, vaso-motor troubles, and special psychic conditions. Hysterical children are precocious, specially with regard to the arts; their affectivities are active, but changeable, the slightest impression produces intense reactions, either joy or grief. Such children have tendencies to lie and dissimulate, their extraordinary statements should seldom be believed, and they often give false witness in courts of justice. Many suffer from mania, are habitually melancholy, and may commit suicide with little or no reason. A commencing hysteria produces the greatest number of cases of simulated organic disease. Hysteria can imitate hooping-cough, enteritis, and meningitis; certain cerebral disorders such as infantile spasmodic hemiplegia, certain medullary affections such as spinal paralysis, nervous disorders such as epilepsy, and organic disorders such as intermittent fever. It is especially in children that neurosis is best simulated. In its mildest form infantile hysteria only appears in psychic phenomena. Children often have pallakinia, tenacious prurigo, and nettle-rash; they sleep badly or lethargically, start and tremble at the slightest noise, and are frightened of the darkness. Their dispositions are flighty, ungoverned, passionate; they are jealous of their brothers and sisters, have ideas unsuited to their age, excessive sympathies and antipathies. They eat badly, are thin, with hollow eyes, and present often attacks of meningism.

Diagnosis.—The diagnosis of hysteria depends on the presence of stigmata; these should be sought with great care, especially in monosymptomatic hysteria. Should they be wanting, the special circumstances causing the accidents and the antecedents of the patient will be of the greatest value in the diagnosis. The possibility of an organic lesion has to be carefully considered. It is impossible to show here the differential diagnosis of hysteria and all the disorders it may simulate; we shall limit ourselves to indicating in the following table the differences between hysteria and epilepsy, the malady most often confounded with it:—

Epilepsy	*Hysteria*
It shows itself during the first fifteen years of life. Generally there have been infantile convulsions.	Although sometimes existing in childhood, it oftenest appears at the time of puberty, and during the first portion of the adult life.
The fits follow night or morning; the patient falls without any previous warning, in consequence there are scars on the face and skull of an epileptic.	The attack follows the fall, and is preceded by an aura. The patient is aware of the attack, and does not injure himself.
The beginning of the fit is marked by an initial scream.	There is no initial scream. The cries come on at a more advanced stage.
During the fit it is seldom necessary to hold the patient energetically, his movements are not prolonged.	The patient's movements are so violent and prolonged that it is often necessary for several people to hold the patient.
The loss of consciousness is absolute. The patient cannot possibly speak.	The patient utters exclamations, and sometimes pronounces sentences. The consciousness comes back during the phase of violent movements.
Emotional attitudes do not exist.	Emotional attitudes exist, as if the patient were acting in a dream.
The delirium after the convulsions is unreasoning and impulsive.	The delirium is rational, and can be directed by speaking to the patient.
At the crisis of the attack the tongue is bitten, there is involuntary defecation and micturition.	There is no injury to the tongue, and no involuntary defecation or micturition.
The general health is deranged by these fits, and the condition is grave and feverish.	The frequency of the attacks do not affect the general health. The temperature is normal.
After the fit, torpor, sleepiness, curvature.	The attack ended, the patient shows no morbid phenomena.
From a clinical point of view, one finds after the fit a rise in the rate of the fixed residuum of the urea, chlorine, sulphates, and phosphates, without inversion of the formula of the latter.	From a clinical point of view one finds a lowering of the rate of the fixed residuum of the urea, chlorine, sulphates, phosphates, with inversion of the formula of the latter.
No hysterical stigmata, no permanent narrowing of the field of vision.	Besides the attack, there are stigmata and permanent narrowing of the field of vision.
Bromides have a therapeutic action most undeniably.	The action of bromides is nil, or inferior to what they are in epilepsy.

Such are the chief points of the diagnosis between these two maladies. With regard to the diagnosis of hysteria, we must bear in mind that the disease may closely simulate a great number of organic diseases. In doubtful cases, even where stigmata may be present, it is necessary to make a distinction between minor symptoms and those which ought to occupy the first rank, namely, the narrowing of the field of vision and anæsthesia.

Treatment

A. *Prophylactic.*—Hysteria is an essentially hereditary malady, and arises from both direct and indirect heredity. Psychic and physical remedies are necessary to counteract the effects of hysteria in an individual so predisposed. Education and physical exercises may do much to correct a tendency to the neurosis. A boy, for example, should be brought up in the family circle, educated by a firm and equable master; all exciting literature forbidden, and physical exercises, such as gymnastics, riding, swimming, and hydrotherapiæ, encouraged, care being taken that the boy does not overdo these things. When he is older the strain of examinations, etc., must be avoided. The girl even more than the boy requires a wise home education; if the mother is highly nervous she should confide her child to the care of a good governess or judicious relatives, for the sight of the mother's nervous outbreaks is bad for the child and favours the neurotic development. At puberty all over-excitement must be avoided. The period of menstruation, with its psychic and physical changes, is fraught with possibilities for young girls. A severe moral shock may make complete psychic and physical repose necessary. All excitement and romances should be forbidden, also excessive dancing, etc.

Among other causes of nervous disorders alcoholic stimulants must be guarded against; subjects should be warned to avoid stimulants, and should not enter any calling that exposes them to the use of intoxicants. With the prophylactic treatment there arises the question, how far marriage and pregnancy are influential in hysteria. It is an old saying that marriage is the cure for this malady, but this is far from being proved. If the marriage is a happy one, and if the young wife is free from care and sorrow, it will certainly have to all appearance a beneficial result. If marriage, on the other hand, is a source of trouble, its influence will be unfortunate. The family ought, then, to be made aware that marriage is not a cure. If hysterical symptoms have not developed before twenty years of age, hysteria is less likely to be serious. The influence of pregnancy is debatable; the moral influences which surround the subject have much more weight than the mere condition itself. Suckling the infant should be forbidden, as it exhausts the mother, and the milk of a healthy wet-nurse is of far more value to the child than that of an excitable mother.

B. *General Treatment.*—Hysteria being essentially a psychic malady ought therefore to have a *psychic treatment.* The first condition of this treatment is for the physician to have entire authority over his patient, that he may know how to speak to him and secure his confidence. He must have sympathy with his patient's sufferings, and be able to assure him of the efficacy of the means used to cure him. It is for this reason that it is often advisable to separate the patient from all outside influences, and place him under strict medical supervision and nursing. In fact it is better that the patient be completely away from relatives and friends. The duration of his absence should vary according to circumstances, and it should be continued for some time after the abatement of the disorder. As the cure progresses the isolation need be less complete, and the patient may gradually resume his ordinary liberty, taking walks, and by degrees returning to his former mode of life. Unfortunately this isolation cannot always be arranged, as even the friends object to it, but grave cases of hysteria can be cured in no other way. Hypnotism is a valuable aid in the treatment of hysteria, but it must not be considered a panacea: numbers of hysterical patients are not hypnotisable. On the other hand, hypnotism itself is not without danger, for repeated *séances* act as a form of attack. It is not impossible that a subject who has been under the influence of hypnotism may fall back into the same state under the influence of suggestion, such as the looking fixedly at some brilliant object. Hypnotism in itself is not a treatment for hysteria, but is an adjunct of the greatest value, which ought to be reserved for special emergencies (convulsions, delirium, paralysis, spasms, etc.). The means followed to produce the hypnotic sleep are, steadily gazing at a bright object, listening to a monotonous noise (the tick-tick of a watch), gentle rubbing of the eyelids, pressure of a hypnogenic zone, and simple suggestion. It is during this sleep that the suggestion may be exercised at the moment when the patient is plunged into the somnambulistic trance. (*See* also "Hypnotism.")

The suggestion to the listening patient deserves to occupy a conspicuous place beside the hypnotic suggestion. The patient benefits greatly by the suggestions of his medical attendant, whether addressed directly to himself or overheard when uttered to some one near him. Very often on hearing the doctor emphatically state the opinion that he is much better and nearly cured, the patient believes it, and half the battle is won. This suggestion is rendered more efficacious by the administration of harmless medicines which the patient believes to be bene-

ficial (bread pills, rye powders, etc.). Methylene blue in pillules, because of the colour which it gives to the urine, is valuable as a means of suggestion of cure. The external treatment of hysteria in general consists of magnets of metals, electrisation, and hydrotherapy, etc. Metallotherapy is based on the belief that the patient has a special sensibility for one or more metals, which, when applied to the surface of the skin, may modify the anæsthesia and the contractures. This therapeutic measure has often been advocated, but the results derived from it are very disappointing.

The electric treatment plays an important and really efficacious part. Static electricity acts like æsthesiogenic and as a tonic; it can be used in the form of electric baths, or if it is better to localise the electricity, it may be used as an electric blast, or brush, or spark. The electric bath is followed by a feeling of comfort and a disappearance of the anæsthesia and the amyosthenia. The appetite improves, and the digestion is stronger. The duration of the *séances* should be from ten to nineteen minutes.

Electrical faradisation, with the aid of a brush, acts most favourably on isolated anæsthesia, or when placed over paralysed parts or contractures. In cases where one cannot have recourse to static electricity on account of the want of proper apparatus, generalised faradisation may be used instead with good effect.

Galvanic electricity is inferior to faradised electricity in the cure of hysteria. It can, however, be used beneficially in cases of local trophic troubles, muscular atrophy, and œdema. Hydrotherapy is of the greatest value on account of its æsthesiogenic action. The cold douche is advantageous in battling with anæmia and general debility, as it favours the absorption and elimination of the therapeutic factors. The best mode of administering the douche is to have it poured on the body at a temperature of 8 to 12 degrees for the space of 19 to 20 seconds, taking care not to project the jet of water on the head or the hysterogenous zones. Sometimes the cold douche cannot be endured, and in such cases one must have recourse to the Scotch douche, either without transition, that is to say, passing suddenly from a temperature of 35° or 40° to a very low temperature, or, if the patient is very sensitive, with gradual transition. In special cases, wrapping in the wet sheet or patting with wet sheet may be beneficial, but these means should, as a rule, merely lead on to the cold douche.

Treatment by mineral waters is not so useful as hydrotherapy; the most highly recommended waters are Neris, Divonne, Bains, Plombières, Luxeuil, La Malou, Wildbad, Ragatz, etc.

Regimen.—The diet ought to be tonic and abundant, but overloading the stomach has to be avoided. In grave cases certain physicians employ a milk diet, as in the Weir-Mitchell cure of neurasthenia, namely, 100 grammes of milk every two hours, together with complete rest and isolation. Ordinarily one does not need to have recourse to this régime, but it is always best to choose light foods and to eat often, say four meals a day.

Medicinal Treatment.—There is no medicinal treatment necessary in ordinary cases of hysteria, the internal medication being purely symptomatic. To calm the patient alkaline bromides may be given, which are far from being as active as in cases of epilepsy. Injecting morphia should be avoided, as there is a risk of hysterical patients becoming morphinomaniacs. The different preparations of valerian (tincture, extract, ammoniated valerian, and preparations of zinc and quinine) have all the great advantage of not being prejudicial. A number of hysterical patients are anæmic, and in their case preparations of iron and arsenic are very valuable. We need do no more than mention the surgical treatment by oophorectomy or hysterectomy. Its success is problematical, and does not compensate for its real inconvenience.

C. *Treatment of the Attacks.*—When the attack is very violent the patient must be guarded from injuring himself. There are two ways of combating the attack itself.

1. In the first rank among the mechanical aids must be placed the compression of the spasmofrenatric zone, which must be searched for in the ovarian and epigastric regions. In order to touch the zone the hand must be pressed into the iliac fossa, and then having overcome the resistance of the abdominal walls, it will be sufficient to place the two first fingers on the presumed seat of the ovary in order to obtain the desired effect. Pressure of the breasts and nape may also be used with advantage. By gently pressing the eyeballs also the effect of hypnotic sleep may be tried; and electric shocks at intervals may arrest the crisis.

2. As regards medicines, the inhaling of ether and chloroform must be used guardedly. In very bad cases chloroform may be tried. Bromide of ethyl and nitrite of amyl have also been used for the same object.

D. *Treatment of Particular Accidents.*—Anæsthesia of the nerves and senses can be subdued by brush electricity and static electricity. Paralysis, however, presents great resistance to the remedies employed, and it is necessary to have recourse to transfer suggestion and massage. In cases of contracture the medical intervention should be rapid, acting upon the hyperæsthetic zone by the aid of static electricity, and on the anæsthetic zone by electric faradisation. Hypnotism rarely succeeds. Movement of the contracted limb must be avoided. Any application of apparatus may aggravate the disorder, and there is the risk of permanently disabling the limb, and fixing the contracture psychically. In certain cases after the con-

tracture has disappeared retraction is observed; surgical treatment of these retractions should not be used, otherwise there may be a return of the contracture. Among the visceral disorders of hysteria it is important to notice anorexia, as it is one which may endanger the life of the subject. Its proper treatment is complete isolation. The feeding tube ought not to be used, except as a last resource, for the patient may become habituated to it. The patient must be forced to eat even by threats, and there must be no yielding as to the kind or quantity of food to be taken.

Nature of Hysteria

The question of the nature of hysteria is singularly complicated and difficult to treat. Although the subject has attracted the attention of numerous writers, it cannot yet be said that everything is clear with regard to the explanation of its phenomena.

The ancients—Democrates, Plato, and Hippocrates—regarded this neurosis as taking origin in the uterus. Hippocrates defined it as "a 'suffocation' of the uterus which rises to the heart, to the liver, to the hypochondrium, to the loins; when it comes to the liver it produces suffocation, intercepting the respiratory path which is in the belly. . . . These symptoms occur especially in spinsters and in widows who, being young and having had children, remain in expectancy. It is best that they become pregnant; as for the maid, she should be advised to marry." In these words the doctrine of the uterine origin of the disease and its treatment by marriage, which has been so strongly adhered to, is formulated for the first time.

Galen, although admitting the possibility of hysteria in the male, associates this malady none the less with disorders of the uterus. He no longer believes, it is true, in the rising of the uterus towards the hypochondrium, but he regards the symptoms of hysteria as dependent on displacements of the uterus (antero-version, retroversion, etc.), and on retention of the menses resulting in a general intoxication of the organism.

Later, during the Middle Ages, the views of Galen were accepted as facts. Cases of hysteria were not uncommon, and the written and illustrated records from this epoch which remain reproduce pictures in which the features of hysteria are readily recognised. Under the influence of predominant religious ideas, the hysterical subject was regarded as a sorcerer or as one possessed of the devil. The stigmata of hysteria were the "signs of the devil," and the sole therapeutic measure employed was exorcism. Thus, far from making progress, the explanation of the phenomena of hysteria became more and more obscure. In the eighteenth century the uterine theory is reverted to. Hoffmann attributed hysteria to a spasmodic contraction of the uterus; nearer the present time, Lonzer Villemag in 1802, and Landouzy in 1846, regarded hysteria as the consequence of uterine trouble.

In our own time certain writers, if they do not admit the uterine, still maintain the sexual origin of the disease. Certain surgeons, few in number it is true, do not hesitate in severe cases of hysteria to remove the ovaries in the female and the testicles in the male. The results of these more than doubtful operations do not give support to the sexual theory. Cases have been recorded where true symptoms of hysteria have developed in nervous females who have undergone such treatment on account of local disease.

The theory which considers hysteria as a nervous disorder which may develop in the male as well as in the female is of relatively recent date. It is to Sennert (1572-1637) and to Charles Lefrois that we owe the conception of hysteria as a disease of the nervous system, a conception still very vague and very erroneous, since the last-named author attributed its symptoms to the presence of a collection of fluid within the head. It is none the less a step forward towards the humoral theory of Sydenham, who has given a perfect clinical description of the disease; for he regarded hysteria as a consequence of debility of the nerves, of impoverishment of the fluids, of a disturbance of the *esprits animaux.*

In the nineteenth century the nervous theory of hysteria gained ground through the works of Georget, Brachet, and Briquetait. "Hysteria," says Brachet, "is a neurosis of the central nervous system, which declares itself more or less suddenly in attacks of general clonic convulsions and by the sensation as if a ball were rising in the œsophagus, at the lower part of which it settles, threatening suffocation." As one sees by this definition, the author has only recognised the convulsive form of hysteria. Through Briquetait the question expanded and the nervous theory evolved. He sought to explain not only the transient convulsive phenomena, but also the persistent symptoms; he showed the part which the emotions play in the disease. "To me," he says, "hysteria is a neurosis of the brain, the symptoms of which consist in perturbation of the vital acts which serve to manifest the affections and the passions." This conception is an advance towards the theory by which hysteria is regarded as a cerebral disease, a disorder of the mind. The credit of showing the important part which is played by the "fixed idea" in the production of the various symptoms of the neurosis, more particularly in traumatic hysteria (paralysis, contractures, etc.), is due to Charcot. He demonstrated the possibility of producing hysterical phenomena by suggestion, and emphasised the existence of a mental state *peculiar* to these patients. He also confirmed from the practical side the theory which regards hysteria as a

mental disorder, by bringing forward the results obtained by moral treatment in the cure of the disease. His colleagues—Marie, Gilles de la Tourette, Sougues, Guinon, Pierre Janet, etc.—have, by their researches, helped to elaborate and explain certain points. While mentioning the name of Charcot, it would be unjust to forget the names of Möbius and Strumpell, who with him place hysteria among the psychoses, putting it among those diseases which they call the diseases by representation (*durch vorstellungen*).

Looking back at the chief stages through which the doctrine as to the nature of hysteria has passed, we see it regarded at first as a disease peculiar to the female and of sexual origin, then slowly coming to be regarded as a disorder of the nervous system, organic by some, functional by others. At the present time the leading authorities on neurology and mental diseases regard Hysteria as a mental disorder. We shall now briefly review this theory.

If hysteria is a mental affection it is not to be included in the group of the true psychoses. The symptoms of hysteria have a *particular* physiognomy which distinguishes them. The writers who have upheld the mental theory attribute the phenomena of hysteria to:—(1) a dual personality, and (2) a shrinking of the field of consciousness.

The theory of the dual personality has been advocated by Möbius. It is a deduction drawn from the *rôle of the fixed idea* (Charcot), in the development of the symptoms of the disease. This duplication of the personality doubtless exists in somnambulism and in delirium, but it is more difficult to explain by the adoption of this theory certain symptoms, such as the anæsthesia and the hyperæsthesia, in which there exists no conscious *activity* of the individual. It is this part of the theory explaining the production of the unconscious fixed idea on which it is necessary to insist. A somnambulist has two personalities, two separate psychological existences, of one of which he is absolutely ignorant. Charcot has shown that there exists a very remarkable similarity between attacks of hysteria and somnambulism.

If then we admit that during an attack the *fixed ideas* are more powerful than in the waking state, it is easy to conceive that these ideas are able to originate in the individual and to develop during a state of unconsciousness. Abnormal states of consciousness are produced, limited, it is true, in what is really an abnormal personality, and it is the existence of this abnormal personality which enables us to understand the development of the symptoms of hysteria.

This theory of the part played by the *fixed idea* is shared by a number of writers. Gurney and Th. Myers hold that hysteria is an unreasonable auto-suggestion under the control of the normal consciousness. If this theory be applied it explains satisfactorily the symptoms of hysteria, but does not explain the stigmata, and for this reason certain writers look for another explanation of the latter phenomena. They admit, however, that these are the consequence of an enfeeblement of the cerebral function.

Féré regards the subject of hysteria as being in a permanent state of psychical fatigue, which manifests itself by a diminution of sensation, of motor power, and of the will.

Oppenheim considers that a condition of irritable weakness and an abnormal excitability, coupled with nervous weakness, play an important part in hysteria.

Jolly admits of an extreme weakness allowing of the exaggeration of the emotions. P. Zanet goes farther than the preceding authors, and tries to determine the nature of this mental debility; he regards it as a defect of attention, attention being maintained with difficulty and easily exhausted.

Whether one considers it from the motor or sensory point of view, it is this want of attention which explains *l'aboulie*, the hesitation which prevents the hysterical subject giving himself up to intellectual pursuits, and at the same time taking in and storing up impressions, and from accomplishing many movements. It may be said that his field of consciousness, on account of its limited extent, can only take in one sensation at a time. This diminution of the field of consciousness prevents the individual elaborating and condensing his thoughts, from assimilating them, from making and transforming them into an integral part of his personality.

Pick of Prague arrived at analogous conclusions when he attributed the persistent symptoms of hysteria to diminished power of attention, of perception, and of movement.

As regards the application of this theory to the explanation of the visceral symptoms: it seems that their mental origin cannot be accepted without question; how does it explain the influence of moral treatment, of isolation, and of symptoms such as anorexia and spasms? Are the persistent vaso-motor phenomena to be regarded as the result of an exaggeration of the *emotions*?

Such, broadly speaking, is the psychical theory of hysteria.

We cannot do better than quote the following formulæ, given by Janet, which form a résumé of the mental theory:—

"Hysteria is a mental affection belonging to the large group of diseases due to cerebral weakness and debility. Its physical symptoms are somewhat indefinite, consisting chiefly in a general diminution of nutrition. It is largely characterised by moral symptoms, the chief of which is an impairment of the faculty of psychological synthesis, an abolition, and a

contraction of the field of consciousness. This manifests itself in a *peculiar* manner and by a certain number of elementary phenomena. Thus sensations and images are no longer perceived, and appear to be blotted out from the individual's perception, a tendency which results in their persistent and complete separation from the personality in some cases and in the formation of many independent groups. These series of psychological facts alternate the one with the other or co-exist. Finally, this synthetic defect favours the formation of certain dependent ideas, which develop complete in themselves, and unattached from the control of the consciousness of the personality. These ideas show themselves in affections possessing very various and unique physical characters."

Recently Sollier has propounded a new theory for hysteria which differs considerably from the conception of the Salpêtrière school. It is based on the well-known fact that hysterical subjects (*hysteria major*) sleep little, and yet do not suffer from fatigue. He concludes that these patients are immersed by day in a pathological sleep, which renders natural sleep impossible. It is for this reason that if one orders hysterical patients who have grave symptoms to wake up completely after hypnosis, a series of phenomena are seen similar to those accompanying the natural waking state, with regression of the personality. To conclude from this that the subjects of hysteria are somnambulists or rather "vigilambulists," is not a wide step.

The state of vigilambulism is modified, not only when one orders the patients to awake, but also when one orders them no longer to be aware of their anæsthetic zones. The return of sensation, obtained in this manner, is sufficient to arrest all the symptoms comprised in the somnambulistic state. This method, which at the same time is a therapeutic agent, is called by the author the "method of arousing the sensibility." These alterations of sensation are, in the same way, dependent upon a torpid condition of the cerebral centres, which are indeed asleep. It is easy to explain by this conception the different symptoms of the neurosis, since, according as such and such a centre becomes torpid, different symptoms will result, and in cases where numerous symptoms are present several centres may be asleep at the same time. This doctrine admits that the existence of cerebral localisation accounts for the dynamic troubles of hysteria. It deserves on account of its originality to be placed alongside of the generally accepted theories.

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Hysteria in Childhood

Hysteria is probably much commoner in children than is generally supposed, and it is a matter of no small importance that its frequency should be recognised, for at this age environment, one of the most potent factors in hysteria, is easily modified and controlled; moreover, education in its widest sense can be carefully planned even in its smallest minutiæ, so as to guard against neurotic tendencies at that period when they are most easily checked.

Age.—There is some doubt as to the earliest age at which hysteria may occur. At least three cases have been recorded in which symptoms attributed to hysteria were present at the age of 18 months, and there have been some very doubtful cases at an even earlier age. Hysterical catalepsy has been seen at 2 years (Mills) and at 3 years (Jacobi), and hysterical analgesia at $2\frac{3}{4}$ years (Barlow). After the age of 4 years hysteria becomes more frequent. The writer has met with hysterical amaurosis with analgesia at 4 years, and has known severe hysterical contracture to occur at $4\frac{1}{2}$ years. But the majority of the cases of hysteria in childhood occur during the five or six years preceding puberty: of 33 consecutive cases under the age of 12 years, which came under the writer's observation, 23 were in children over the age of 8 years.

Sex plays a far less important part in the hysteria of childhood than in that of adults. The predominance of the female sex which is so striking in adult hysteria is much less noticeable in this condition in children. While the proportion of males to females in adult hysteria is variously stated at 1 to 3 (Pitres), 1 to 10 (Bodenstein), 1 to 20 (Briquet), in children it appears to be about 1 to 1·25.

In the series of 33 children mentioned above, 18 were girls, 15 were boys. It would seem, however, that when boys are affected they are liable rather to the milder manifestations of hysteria, than to the severe forms often seen in girls.

Predisposing and Exciting Causes.—There can be little doubt that in many, perhaps in most, cases the tendency to hysteria is congenital—the child is born with a neuropathic tendency; and in this sense the dictum of

Pitres, "on nait hystérique, on ne le devient pas," is probably correct.

The influence of heredity is perhaps even more marked in the hysteria of childhood than in that of adults, and it seems likely that the appearance of hysteria at a very early age may be determined in some degree by the intensity of the neurotic inheritance. Not only hysteria itself, but epilepsy, migraine, and other neuroses in the parents predispose to hysteria in the child. Alcoholism in the parents seems to be an important factor.

But it is often difficult to discriminate between the results of heredity and the results of environment. Childhood is pre-eminently an impressionable age, and the child who is in constant association with an hysterical mother, or spends its early years in frequent fear of a drunken parent, may perhaps develop hysteria apart from any influence of heredity. Moreover, such parents are as a rule but poorly qualified to encourage in their children that control of the will, by the intellect rather than by the emotions, which should be acquired during early life, and the lack of which appears to be at the root of some at least of the manifestations of hysteria.

The frequency of rheumatic heredity is also very noticeable. Apart from chorea, the children of rheumatic families seem to be specially prone to minor functional neuroses, and in a considerable number of the cases of hysteria in childhood there is a family history of rheumatism.

A tendency to hysteria is often fostered by the atmosphere of home life. In particular the solitary child who has to pass its early years in the society of adults, and scarcely ever associates with other children, not only becomes self-centred, and acquires those habits of introspection which are so conducive to hysteria, but too often suffers from excessive attention; its trivial ailments and injuries receive an exaggerated sympathy, and its precocity is a constant theme of admiring approval.

The influence of school life as an exciting cause of hysteria is a point of considerable practical importance. It has happened repeatedly that the onset of the hysterical condition in a child has occurred just at the time when school examinations were at hand, and there is abundant evidence that the pressure of school work, especially the keen competition of examinations in the present day, does act deleteriously upon children of nervous temperament. Chorea, somnambulism, fæcal incontinence, and migraine are certainly in some cases closely related to the mental strain of school work, and there can be little doubt that school work, in the same way, may be the exciting cause of hysteria.

But apart from the mental strain, there are some children upon whom school has a harmful effect for an entirely different reason. These are the extremely timid children to whom school is a daily misery, not because they dislike or are unable to do the work, but because they cannot shake off a fear of the teacher, or in some cases a morbid shyness, which makes all association with strangers, even with classmates, a trial to them.

School punishments also, especially corporal punishments, have been the exciting cause of hysteria and other functional neuroses in childhood; such severe symptoms as hysterical convulsions have been caused in this way.

It is most important in this connection that it should be realised that one of the earliest manifestations of the hysterical temperament in children is sometimes a condition closely resembling moral insanity. The child is volatile and passionate, perhaps rude or even vicious, and may be almost unmanageable; as such it is in continual disgrace, and is apt to be severely punished. In these children injudicious punishment does nothing but harm, and may bring on a troublesome outbreak of hysteria.

Fright is an occasional exciting cause of hysteria; and this is hardly to be wondered at, for the effects of fright on young children are often very profound, and the wilful frightening of children, whether in punishment or in jest, is a cruelty which cannot be too strongly condemned.

Sensationalism is also an exciting cause of hysteria in children. The thrilling ghost story, the still more unwholesome spiritualistic *séance*, which in one case recorded by Charcot produced severe hysterical symptoms in three children in one family—such things are full of danger for the child with hysterical tendencies. And one cannot pass over the influence of sensational religionism—a very different thing, be it observed, from religion; no small number of cases of hysteria in childhood have occurred in schools, especially convent-schools, or other institutions in which the children were exposed to this unwholesome influence.

Traumatism plays quite as prominent a part in the hysteria of childhood as in that of adults. In the best authenticated case of hysteria at the age of 18 months which has been recorded (Gillette), transient hysterical paralysis of both arms was apparently due to a very slight traumatism of one arm.

Depressing influences and possibly toxic conditions are also occasional causes of hysteria in children. In one child under the writer's care the strain of watching by a sister's deathbed was followed by intermittent hysterical spasm of some of the cervical muscles, and in several cases the onset of hysteria followed immediately after influenza.

Some writers have laid much stress upon masturbation as a cause of hysteria in childhood, and it seems likely that the concomitant excitement and subsequent depression may aggravate hysterical tendencies; but it has to

be remembered that masturbation and the grosser forms of immorality which are occasionally met with in children are certainly in some cases, perhaps in most, the manifestation of the neuropathic tendency rather than its cause.

Imitation certainly plays a large part in the hysteria of childhood. As far back as the thirteenth century epidemics of hysterical symptoms occurred, affecting children as well as adults, and sometimes only children. Quite recently epidemics of hysterical convulsions and other symptoms have occurred in schools; and there can be little doubt that this imitation-hysteria is not all mere shamming; auto-suggestion, prompted by the example before the child, probably induces a genuine hysterical condition; at the same time malingering is by no means unknown in childhood.

Symptoms.—It is unnecessary to repeat the general symptoms of hysteria here, for they have been described already in the article on Hysteria in Adults, and there is scarcely a symptom of the adult affection which has not been observed also in children. There are, however, some special features of this condition, as it occurs in childhood, which are worthy of notice.

The early years of the boy or girl who in later life develops hysteria, not infrequently show certain indications of the neuropathic taint. When the hysterical symptoms occur it is often easy enough to see these prodromal indications in retrospect; but for the child's sake it is most important that their significance should be recognised earlier. Certainly from the age of 2 or 3 years these early indications may be noticeable, and it may be that even in infancy the occurrence of infantile convulsions may sometimes, as Dr. Coutts has suggested, foreshadow the neurotic tendencies of later life. Such children are often extremely passionate; from caressing affection they will pass suddenly, if thwarted, to a perfect fury of passion. In other respects also they are extremely volatile, and their behaviour, without being insane, is often unreasonable, and they are in consequence extremely difficult children to manage. Some of them also show excessive timidity or a morbid shyness. They are troublesome too in their feeding, full of fads, "can't eat this," and "can't eat that." They are apt to exaggerate their ailments. Often they are solitary children who do not care to associate with other children, and show no healthy interests in the games and amusements of childhood. As has been already mentioned, sexual immorality is sometimes an early manifestation of the neuropathic tendency, and some of these children, before the appearance of definite hysterical symptoms, have been so vicious in their habits that they have been expelled from schools as unfit to associate with other children.

As regards the hysterical attack, while in adults it often manifests itself as a complicated grouping of symptoms, in a child it tends to be much simpler. In many cases only one or two symptoms are present, and in the writer's experience motor symptoms — and of these the spasmodic rather than the paralytic — have seemed to be much commoner than sensory.

Localised hysterical spasm is one of the commonest manifestations of hysteria in childhood; in one child there is a sudden jerk of the head to one side, in another a transient spastic condition, shifting at intervals of a few hours from limb to limb, in a third a continuous muscular spasm drawing the foot into a position of talipes, and not very rarely the muscles of phonation and of respiration are affected, and a frequent short, dry, sometimes barking cough or some other curious sound is produced.

In children these spasmodic affections are often rhythmical. In two boys aged 10 years, fine tremors of the whole body occurred, and in one case could easily be produced by suggestion; in another case, a girl, similar fine clonic spasm was limited to the lower jaw; in another girl of 11 years, there was rhythmical belching with a curious gurgling sound, as if air were bubbling down the œsophagus; the symptoms ceased directly the child was put in the horizontal position; the sound was so disturbing that the child had been unable to attend school. Hysterical convulsions, however, the hystero-epilepsy and clownism of major hysteria, are much less common in children than in adults; in 4 of the 33 cases mentioned above, hysterical convulsions were present, preceded in one girl, aged 11 years, by the globus hystericus. Sometimes, as in adults, these attacks can be brought on or stopped by pressure on certain areas, the so-called hysterogenic zones, but these are very rare in children. The writer met with one case of hysterical dyspnœa in a girl aged 8 years, in whom the attacks could be stopped by pressure on the umbilical region.

Of sensory symptoms hyperæsthesia is probably the most common in childhood; anæsthesia and analgesia are certainly rare. The limitation of the field of vision, to which so much importance has been attached as a symptom of hysteria, is occasionally found in children, but it is so difficult to test a child's vision with the perimeter that such observations are apt to be fallacious. Hysterical blindness and deafness have occurred but rarely in childhood. Complaint of headache is not uncommon in association with hysterical symptoms in children; and in some cases its daily recurrence at a certain hour, or its disappearance when the child's attention is distracted, or its curiously localised character, or the presence of hyperæsthesia of the scalp, suggests that the headache is really hysterical.

A very important group of cases is that in

which the hysterical symptoms closely simulate organic disease, the neuromimesis of Paget. Joint symptoms are perhaps the commonest of these in children. The hip-joint in particular is often affected, and it is sometimes very difficult to differentiate hysterical coxalgia from rheumatic or early tubercular disease of the hip. It may indeed be almost impossible to exclude rheumatism; the hip-joint is one of the favourite sites for rheumatism in childhood, and the objective evidence of articular rheumatism in childhood is often extremely slight; moreover, as already mentioned, hysteria is specially liable to occur in the children of rheumatic families; and, lastly, what began as a slight attack of genuine rheumatism may persist as hysterical coxalgia.

Lateral curvature, early spinal caries, and talipes are all simulated by hysteria, but in these, as in the joint cases, the nature of the trouble is often shown by the sudden onset of the symptoms, the diffuse character of the tenderness over the supposed seat of the disease, the variation or disappearance of the symptoms when the child's attention is distracted, the presence of other symptoms of hysteria, and the rapidity of cure by such "moral treatment" as is effectual in hysteria.

A very puzzling condition is that which has been called "pseudo-meningitis hysterica." Several instances of this in childhood have been recorded. The following case may serve as a typical instance. A girl aged 9 years had influenza. The temperature was raised to 101° or 102°, but became normal after a few days. From that time she was drowsy, with obstinate constipation, vomited once, and complained of continual headache in the right frontal region; at one time there was photophobia; and there was great difficulty in persuading the child to take food. The child was apathetic, scarcely ever spoke unless questioned, and just before the writer saw her the pulse was reported to have fallen to 45, and the respiration had been of the Cheyne-Stokes variety. The child was thin and extremely weak; she lay curled up on her side, with the face turned away from the light. The heart acted irregularly, the abdomen was much retracted. There was no irritability, the child answered questions readily, there was no stiffness of the neck and no affection of cranial nerves, the discs were normal. Meningitis was suspected, but a few days later analgesia appeared below the knees, and on being removed to hospital and treated with electricity the child was well in about ten days.

Diagnosis.—For the general diagnosis the reader is referred to the previous article on Hysteria. The diagnosis of hysteria in childhood must be approached with the utmost wariness. The fact that no obvious cause can be found for aches or pains or other symptoms is not sufficient reason for labelling them hysteria. Where there is any doubt it is safer as a rule to assume that the condition is not hysterical than to risk the serious error of supposing a case of organic disease to be one of hysteria. Hysteria, moreover, is sometimes associated with or started by an organic lesion; hysterical joint affection, for instance, is sometimes started by a slight articular rheumatism.

The difficulty of diagnosing hysteria in children is further increased in some cases by the fact that only one symptom is present; the so-called "stigmata" of hysteria are commonly absent.

Prognosis. — The immediate prognosis is probably better in children than in adults. It is very rare for hysteria in childhood to resist treatment obstinately as in later years; and as a rule the symptoms disappear in a few days, or at most in a few weeks, under suitable treatment. But the neuropathic tendency remains, and therefore relapses, usually perhaps with some new symptoms, are apt to occur. Moreover, a study of severe cases of hysteria in adult life shows that in many such cases there have been symptoms of hysteria in childhood, and on this account the prognosis as to the future must be guarded, for in some cases at least, hysteria in a child is the indication of a strong neuropathic taint, which is likely to show itself in graver manifestations in later life.

Treatment.—*Prophylactic.*—There are few conditions in childhood which call for more experience and tact in management than the early manifestations of the hysterical tendency; and by this is meant not the fully developed hysteria which is commonly recognised as such, but that less-defined group of prodromal symptoms, noticeable almost from infancy, to which attention has been drawn above. Home life, companions, amusements, education, every detail of the child's life, must be carefully supervised, so as to avoid those harmful influences which have been mentioned already as predisposing and exciting causes of hysteria. The choice of a nurse is a matter of the utmost importance. To deal with such children successfully requires more than ordinary tact in the nursery; they need a nurse who is quiet and firm, who knows when to take notice and when not to take notice, when to sympathise and when not, and, above all, one who never loses her patience.

In most cases it is probably wisest to send such a child to school as soon as it is old enough, rather than to have a governess or tutor at home; for the mixing with other children helps to counteract the tendency to become self-centred, which is often noticeable in these children, and, moreover, gives them a wider range of interests. But the conditions of school life must be properly regulated; anything like brain-worry—and this is possible at a very early age — must be carefully avoided. The best beginning is undoubtedly the kinder-

garten, and for a child of 4 years, an hour and a half in the morning may be quite sufficient. The teacher should be informed of the child's peculiarities, and it is essential that a school should be found where the teacher has the necessary tact and patience to avoid scolding and punishment, which are worse than useless for these children.

In these, even more than in other children, education must include not merely mental, but also physical training, whether it be by a well-regulated gymnasium, or by Swedish drill, or by dancing, or by some other form of disciplined exercise.

The amusements and interests of the child must not be neglected, for they undoubtedly exercise a considerable influence on the development of hysteria; like the "idle hands," the idle mind is likely to come to mischief, and it is for those who have the care of these children to encourage in them a healthy interest in games, animal pets, the tending of flowers, simple botany, entomology, or some such occupation.

When the home environment is so unfavourable that judicious management cannot be expected there, it is sometimes advisable to send the child away altogether to more wholesome surroundings.

The general health must be carefully considered, for any depressing cause favours the onset of hysteria; the tendency to vicious habits must be remembered; masturbation in particular, an early indication of the neuropathic taint in girls as well as in boys, must be checked, for it may precipitate the onset of hysterical symptoms.

Therapeutic.—The treatment of hysterical symptoms in childhood is the same in principle as in adult life, and it is unnecessary to repeat it in detail here. Change of environment is one of the most powerful remedial measures. Many children speedily recover when removed to hospital or to some place where they are among strangers and strange surroundings. A change from town life to the varied interests of sea-side or of farm life is often helpful, and such measures may be more effectual than drugs.

Cold douching, massage, and electricity are of value in particular cases.

The influence of suggestion is probably more often of use in children than in adults; an impressive assurance that the symptoms will speedily disappear considerably assists recovery. Moreover, in children, such aids as promises of pleasures conditional on the disappearance of the symptoms are more often available, and are very valuable in treatment.

The use of hypnotic suggestion for children suffering from hysteria has been advocated by Bérillon, but some observers have stated that mental disturbance has followed its use, and on the whole it may be doubted whether it is ever advisable to make use of hypnotism for a condition which, in childhood, is probably always curable by other methods. It may, however, be useful for those children in whom vicious habits, especially masturbation, cannot be checked in any other way.

Hysteria, Surgical Aspects of

Hysteria from the surgical point of view, and as I understand it, is that condition in which there is imagination, imitation, or exaggeration of pathological affections. It occurs mostly in females and persons of nervous temperament, and is due to some nervous derangement which may or may not be pathological. Hysterical affections often closely resemble pathological conditions, the explanation of this not being always easy. Sydenham says, as quoted by Holmes, "In whatever part of the body it be seated, it immediately produces such symptoms as are peculiar thereto." Mimicry will not explain the resemblance in every case. Some nervous diseases may closely resemble hysteria. I remember well one case in which hysteria was diagnosed by at least five doctors, including myself, and in which an intracranial sarcoma was discovered post-mortem. Again, hysteria has to be differentiated from malingering and fraudulent shamming. Hysteria is not the same as hypochondriasis; neither is it a form of insanity, though in some cases it may be difficult to draw the line. The term "hysteria," in the sense in which it is now employed, is not, strictly speaking, correct. Sir James Paget suggested "neuromimesis," or "mimicry," and Mr. Holmes uses the word "simulation." Both are good expressions, but neither describes fully the various conditions that are classed together as hysterical.[1] After all, the very indefiniteness of the terms "hysteria" and "hysterical" makes them the most suitable to use, at least until some better expression is invented.

Desiring to make the following remarks as practical and useful as I can, and realising from my own experience that the initial, and I may say main difficulty in cases of hysteria, is the diagnosis, I shall treat my subject mainly from that point of view. Is it not the case that in every instance of hysterical spine, or joint affection, the difficulty is to make sure that there is not present some real organic disease?[2]

[1] Brodie included them all under the term "neuralgia of joints."

[2] Paget says that it is more difficult to diagnose and cure hysterical affections than actual disease.

Any one who wishes to enjoy a masterly description of the subject should read Sir James Paget's clinical lectures on Mimicry. In what I am now writing I shall follow these lectures somewhat closely, and will refer to them frequently.

In any given case, according to Paget, we have three things to investigate—the etiology, the nervous history and condition of the patient, and the condition of the part affected. He urges that the last is the most important, and most to be relied upon in forming a diagnosis.

Let me suppose, then, a case of hysterical joint affection. The patient comes, or is brought to us, supposed to be suffering from joint disease. There will probably be a history of some injury, or of some mental shock, or the affection may have come on gradually. We may also elicit some information that, taken in relation with the local conditions, may throw light on the case. For instance, an injury may have occurred so long before, that, had actual disease existed all that time, changes would be evident in the joint of a very marked and easily recognised character. The condition of the joint, however, may be quite different. And the want of due relation between time and symptoms may show that no real disease is present.[1]

Valuable evidence may be obtained by knowing that the affection came on after some mental shock, fright, worry, disappointment, debilitating fever (Jolly), overwork, or some such nervous perturbation.

Reliable data may be obtained from the personal and family history. A full treatment of these will be found in the general article (p. 308). I shall merely say here that a neurotic or alcoholic personal and family history will make hysteria more probable, while on the other hand a gouty, rheumatic, syphilitic, or tuberculous history would make one more cautious in coming to such a diagnosis.

According to Paget the patients are mostly females, of nervous temperament, easily fatigued, anæmic, liable to flushes, sensitive to pain, or anæsthetic. They think of themselves constantly and of the part affected; "egotism has its keenest life at and about the supposed seat of disease." They are "content, almost happy, in their afflictions," or they are discontented, and make every one miserable around them. They are fond of telling every one of their troubles, and thus court sympathy, for which they have a morbid craving. Will power is deficient in one direction, though some have very strong wills when their interests are concerned. "I cannot" looks like "I will not," but is "I cannot will" (Paget). The family history will disclose a heredity of insanity, epilepsy, intemperance, or some other nervous disorder.

All these points will have been made out while we are examining the affected part, and, if the case be one of simulation or mimicry—a hysterical joint—we will find the following leading characteristics or symptoms present:—

1. *Pain.*—The pain complained of will be in excess of what we usually find in the disease that is simulated. The pain will also be superficial, rather than deep, gentle pressure and pinching the skin causing as much or even more expression of pain than deep, firm pressure. The pain will also vary at different times,[2] may shift from place to place, and may occur in places that we do not usually find painful in actual disease. Pain may be associated with or may alternate with anæsthesia. It seldom, if ever, interferes with sleep.

In joints the forcible approximation of osseous surfaces does not cause pain if carefully performed, and there is no starting at night of the typical kind.

2. *Temperature.*—The general temperature is never raised in the same way as by disease. If there be any rise it will be irregular, capricious, or perhaps fictitious. It is more common to have the general temperature subnormal. If the temperature be above normal the patient should be examined all over. There may be some disease elsewhere causing the rise (Paget).

The same may be said of the local temperature. It is generally lower than normal, the affected part being blue or dead white. If there be any rise of temperature it will be associated with a flushing of the skin and quite temporary.

Paget dwells strongly on the diagnostic importance of the peculiarities of temperature, and points out the risk of being misled by this flushing. He considers that in highly nervous persons any part of the body may flush, just as the face does, from special attention being drawn and directed to it. He considers also that a transient gouty condition may give colour and heat to a hysterical joint. These points have to be made out by the surgeon while he is examining the joint—passing the hand over it, gently moving it, closely scrutinising it, and at the same time questioning the patient, both with the object of diverting her attention from the part being examined, and of eliciting information as to the patient's history, constitution, heredity, and other subsidiary symptoms.

In carrying out these manipulations another symptom will come into prominence:—

3. *Rigidity.*—Stiffness or rigidity of the joint may be present to an excessive degree, in the flexed position if the hip or elbow be affected, in the straight position if the knee. On the other hand, but less frequently, the limb may be limp and useless, the patient complaining of weakness rather than of pain—free movement being possible—such as would not occur in disease. Rigidity is more common and is usually exces-

[1] Holmes says "there is no commensurate impairment of health."

[2] Pain may remit, or intermit and recur suddenly (Jolly).

sive, that is to say, greater than is met with in actual disease, all movement being strongly resisted. Sometimes it is possible to see the patient stiffening the limb in anticipation of the surgeon's manipulations. In such cases, if the patient's attention be diverted by conversation, the limb may be gently yet freely moved.

In this connection I may refer to the position which the limb may assume. Paget says, "Subjective intensity of pain brings about the same postures as are assumed instinctively for the relief of pain." He also points out that there may be unwilling or unconscious imitation. This we should always bear in mind.

Barker says that the limb assumes the position commonly observed in disease. This, however, is not always the case.

4. *Swelling.*—Swelling is said to be conspicuous by its absence. My own experience is that there is generally an *appearance* of swelling of the affected joint and also of wasting of the muscles of the limb. But both these conditions are more apparent than real in hysterical affections.

Swelling of a joint is made more apparent by muscle wasting, as in other affections. Careful measurements and comparisons with the other limb will demonstrate that the joint is practically the same as the other. There may be some puffy swelling, but that is generally slight. Wasting of the limb, however, is usually present to a certain extent. This is only what one would expect from the limb not being used as much as the other one. But there is something more that has to be taken into consideration. There seems to be a possible simulation of atrophy, a hysterical shrinkage (if one may use the term) that occurs, and is very apt to mislead. This disappears under the influence of an anæsthetic.

A hysterical limb may, however, be quite plump, with a good layer of subcutaneous fat. Such a state of matters should make one suspicious. But I have, on the other hand, seen cases of tuberculous disease in which there was a fair amount of fat on the affected limb.

Lastly, one may meet with hysterical œdema; that is to say, fictitious œdema that may disappear under an anæsthetic, and is not therefore due to disease. Charcot describes this hysterical œdema. I have myself seen it in a case which I published in *The Hospital* in 1894.

Now let us still suppose that we have our patient before us. We have carefully examined the case, especially locally, tested the pain, the temperature, the rigidity, the apparent swelling and atrophy. If we still have any doubt we should employ a general anæsthetic. There may be some incipient or latent affection, the symptoms of which are very slight, or perhaps masked by the hysterical condition of the patient.

Suppose the patient to be under the influence of chloroform. If the symptoms resembling disease gradually disappear, and as gradually reappear as the patient comes out, then we may presume that the case is hysterical. If, on the other hand, we discover some evidence of disease, such as grating, we can be quite sure that disease is present.

One should not forget that in a hysterical patient there may be great exaggeration of a symptom, such as pain. This is not a case of hysterical joint, but of joint disease in a hysterical patient, and has to be treated as such. We should also remember that a long continuance of hysterical conditions may bring about actual disease. Mr. Holmes says that the influence of the mind on the body is so powerful that it "may lay the foundation of organic disease."

Before leaving this subject of diagnosis let me say that there is one thing, which I cannot call a symptom, very characteristic of hysterical affections, and on which I have very much relied in forming my diagnosis. It cannot be put in one word. It is the presence, more frequently the absence, of some symptom which would not be absent or present in the disease which is being simulated. In other words, the symptoms are not according to type, they are not convincing, they have not the ring of truth about them. One's suspicion is aroused. Something is here that should not be, or something is awanting that ought to be here. Paget says, "The absence of symptoms should be weightier than the presence." Mr. Holmes says that the local symptoms show contradiction or inconsistency. Another way of putting the matter is, in the graphic words of Paget, "There are sensation signs of disease without a single mark of any organic change produced by them." That is to say, you don't find grating, unnatural mobility, osseous thickening, pulpy synovial membrane, actual swelling, or such like conditions which, if present, would prove decidedly diagnostic of disease.[1]

Now, at the risk of repetition, I must put the case again shortly. To reach a correct diagnosis the surgeon must examine the joint (or other part) carefully, with an unbiassed mind, and with the object distinctly before him of *discovering disease if it exists.* If, under these conditions, he is not convinced that disease is present, then he may conclude that the condition is hysterical. If, on the other hand, he finds the slightest trace of disease, or even is unable to say certainly that no disease exists, it is safer to treat the case as one of real disease. Diagnosis should, however, not be uncertain in the vast majority of cases.

I have not yet spoken of the connection between hysterical affections and the sexual

[1] Hysteria may be produced by some local affection. An example was published in the *British Medical Journal*, 28th July 1900, in which the irritant was an ingrowing toe nail. This is not the surgical hysteria of which I am writing. There is no simulation of disease. Any other peripheral irritation might produce the same effect.

functions. In the female you may find an enlarged or sensitive ovary, or the symptoms of which she complains may vary with the menstrual periods.

In the male you may have an undescended or partially descended testis or a phymosis. In either sex there may be the practice of masturbation. Dr. Jolly says that "irritative pathological conditions of the genitals," which he defines as "sexual abstinence or over-irritation," are often present in hysterical patients. These give rise to "diseased reflex actions" on account of the "increased irritability of the sensory system."

This connection between the sexual functions and hysterical affections does not, in my experience, always exist.

Hysteria of the Spine.—This subject will be dealt with also under the heads of spinal affections, orthopædics, etc.

I would merely say on the general subject that diagnosis is often very difficult, and that therefore a careful and thorough examination is always necessary. It is not difficult to overlook a slight rigidity of the spine, or a little difference in the length of the lower limbs.

I shall not say anything about lateral curvature, as I am sure it will be fully dealt with elsewhere. What I have got to say will be limited to the diagnosis of hysterical conditions from Pott's disease of the spine.

We may further limit our subject to the earliest stage of caries before any prominence has formed, because the advanced stages of this disease cannot be simulated.

In forming a diagnosis the most important symptoms to depend upon are pain and rigidity.

In Pott's disease the pain is produced by firm pressure on the spinous processes of the affected vertebræ, and the same pain is produced by downward pressure on the shoulders or head. In more advanced cases you have the patient supporting the head with his hand, or lifting the weight off his back by placing his hands on the chair or bed when sitting up.

In hysteria, on the other hand, we have the pain (as we have seen) excessive, superficial, varying, and not caused by downward pressure, if this be made carefully and without the patient knowing why it is done.

As regards rigidity there is an equally well-marked distinction. In caries you have this symptom always present. It is made out by causing the patient to flex, extend, and rotate the spine, when some stiffness in movement, or actual fixation of the diseased portion of the neck or back, will be at once made evident.

In hysteria, on the other hand, the back is more frequently limp and weak. Rigidity if present is likely to be spasmodic and of the nature of opisthotonos.

There is one thing that I think should always be borne in mind, viz. that in many persons there is a sensitive area in the spine that has no relation with disease in any way whatever.

Girdle pain is not an infallible proof of caries of the spine. It can be imitated. But as this symptom is not usually an early one in Pott's disease the absence of a projection and of other symptoms will show that the case is one of simulation.

One must remember that severe spinal pain, somewhat hysterical in character, may be caused by a malignant tumour or aneurysm. Neither must it be forgotten that we may have caries occurring in a hysterical subject.

Hysterical Affections of Joints.—The diseases which are apt to be simulated are tuberculous disease of the hip and knee, Brodie's abscess, loose cartilage in the knee, and chronic synovitis (of adhesions, contractures, and deformities I will speak afterwards).

The Hip.—The symptoms that will be imitated are pain, lameness, flexion of the joint, lordosis, lateral curvature, flattening of the hip, and adduction with apparent shortening of the limb. As in the case of Pott's disease it is only the early stage than can be simulated.

Let us imagine a case. The patient, who is usually delicate-looking and anæmic, limps or is carried in, or we may have to visit her lying in bed. On examining we may find all the symptoms above enumerated apparent. On careful investigation, however, we will find that the pain is, as usual, exaggerated, superficial, and not increased by pressure on the trochanter, heel, or knee. All deformity can be made to disappear under careful and gentle manipulation. The normal movements of the joint may also be obtained if performed cautiously and when the patient's attention is otherwise occupied. If the patient be a female the iliac fossa should be examined for a sensitive ovary, especially if there be well-marked flexion of the hip with lordosis. A very good plan, especially in young persons, is to examine the healthy limb first. In doing this the forcible flexion test for hip disease may be employed with advantage. If the healthy limb can be thoroughly flexed on to the abdomen without raising the affected limb, the latter cannot be diseased. The patient may then be made to stand and walk, and to stand on one leg—first on the sound one, and then on the affected one.

If the patient be a boy he should be examined for phymosis and hernia. I have had two cases of complete cure after circumcision. If there should still be some doubt the patient should be placed under the influence of an anæsthetic, and the conditions of the joint carefully and thoroughly examined. There are reasons why an examination under an anæsthetic may be specially necessary in the case of females.

The Knee.—Tuberculous disease or chronic synovitis are most frequently simulated. The

patient limps badly, or refuses to use the limb at all. There may be the history of an injury —perhaps months or even years before. Pain is said to be very severe, especially on movement. Examination should be made with both limbs bare so that the surgeon may be able to compare them. At first a difference between the two may be apparent, but closer examination will show this to be slight if at all.

On looking for painful spots these will be found to be, as usual, superficial, variable, and uncertain. By this the possibility of a Brodie's abscess may be often at once eliminated. Anæsthetic areas also may be made out. It will generally be found that the pain does not prevent sleep, and that there is no starting at night. This last symptom may be simulated if the patient has heard or read about it. The temperature of the joint will be normal or subnormal, the skin being sometimes blue or dead white in appearance. The joint may seem to be swollen, but there will be no fluctuation, or thickening of the synovial membrane. The bones will be normal, and the patella will be movable, but not floating. The relation of the bones to one another will also be normal. There may be some "puffiness" or even œdema, and perhaps a transient redness — as has already been pointed out.

If the patient be otherwise well nourished a comfortable layer of subcutaneous fat will be easily made out.

On the other hand, atrophy of the limb may exist to a certain extent from want of use, and may be increased by what I have ventured to call "hysterical shrinkage." Careful measurements will, however, demonstrate that the joint is not enlarged, and that the muscle wasting is more apparent than real. The position which the limb usually adopts is straight, but the joint may be flexed. The limb is usually rigid with the muscles firmly contracted so as to prevent motion, which is said to be very painful. If the surgeon manipulate carefully, however, he may be able to satisfy himself that mobility is present and is perfectly normal and painless.

In some hysterical cases, instead of pain with rigidity, the patient complains of weakness. She does not use the limb, and, in consequence, there may be considerable atrophy. One very important point, on which Paget lays considerable stress, is, that the apparent disease is not so advanced as would be the case had actual disease existed for the period during which the patient has complained.

Sometimes swelling and redness may be caused by irritating applications. If this be done fraudulently it can generally be discovered by investigation.

One must be careful not to overlook some chronic or subacute affection, such as gout or rheumatism.

The symptoms of a loose body or cartilage in the knee joint may be simulated. Generally, however, the pain complained of is excessive and uncertain in character.

Lastly, under an anæsthetic all symptoms disappear, and as the consciousness returns so do the symptoms. This generally settles the diagnosis.

Hysterical Affections of Muscles.—These are often pure examples of mimicry. The patient sees some one affected with the genuine disease, and, consciously or unconsciously, voluntarily or involuntarily, imitates the movements. As examples I shall refer to only two affections, spasmodic wry-neck and squint.

Wry-neck, if hysterical, comes on suddenly, varies in degree, may intermit, and may pass from the one side to the other. It disappears entirely during sleep. It may be connected with intestinal irritation. It tends to disappear, and may do so suddenly.

This will be more fully discussed under "Wry-neck" and "Spasmodic Affections of Muscle." (*See also* "Strabismus.")

Hysterical Tumours.—Abdominal phantom tumours, as they are called, seldom come into the hands of the surgeon. They generally go to the physician and obstetrician. I have little to say of them beyond that they disappear when the patient is placed under the influence of an anæsthetic.

Mamma.—Painful affections of the breast are sometimes supposed by patients to be malignant tumours. If the non-existence of a tumour be made out the patient is generally much comforted. If, however, she be hysterical, she may prefer her imaginary tumour and may disbelieve the surgeon's comforting assurances. This is a very characteristic symptom. In regard to such cases Paget says that we must judge by what we feel, and not by what the patient feels or says she feels.

The pain complained of in hysterical breast is, as usual, excessive and varying; sometimes passing from one breast to another. I should like to say in this connection, that in my experience the removal of the breast for neuralgia (hysterical?) when no actual disease can be made out is most unsatisfactory. The patient may be better for a time, but the pain returns in the other breast, and if that be removed, as I have seen done, the pain is transferred, after a time, to some other part.

Hysterical Intestinal Obstruction.—Hysterical subjects are well known to be prone to constipation. They are neglectful of the calls of nature, and are sometimes even dirty in their habits. Under such circumstances it is possible for the bowels to become torpid, and loaded with fæces to such an extent as to cause obstruction. Such cases differ in no respect from ordinary examples of fæculent obstruction as regards either symptoms or treatment.

But there is another class of cases in which ob-

struction seems to occur from much the same cause as retention of urine in hysterical females. The patient seems to exercise an inhibitory influence over the intestinal peristalsis, with the result that there is constipation and also sickness. In my experience the diagnostic point is that the sickness has not the well-known characteristics of obstruction vomiting, and is never fæculent. On the other hand a powerful purgative, which would be dangerous in a real case of obstruction, is usually quite successful in overcoming the patient's inhibition of her peristalsis, and in getting rid of the obstruction.

Massage has also been successful in such cases.

Hysterical Contractures.—While these are described in the general article (p. 314) it may be advisable to refer briefly to their surgical bearings.

Such cases, in my experience, are not usually purely hysterical. There is the history of an injury or of some joint affection. The case has, however, become hysterical because the dominant factor standing in the way of recovery is the unwillingness of the patient to make the necessary efforts, or to use the necessary means for recovery.

Let me put the matter another way. In making our diagnosis we have to settle whether the case is one of pure hysteria, or of hysteria affecting a joint or group of muscles that once were inflamed, or of hysteria grafted on to a pathological condition the result of some previous affection.

To arrive at a correct diagnosis in such cases it is generally advisable to place the patient under the influence of an anæsthetic.

Before deciding we must investigate into all possible causes of contracture, such as joint disease, nervous affections, muscle affections and adhesions.

We must not forget that permanent muscle changes may result from long-continued hysterical contractures. Jolly says that contractures may occur after a hysterical convulsion.

Under an anæsthetic any adhesions will usually be both diagnosed and broken down easily.

Treatment.—What I have to say on the subject from the surgical point of view must be necessarily very brief. Paget says that we have three things to treat—the constitution, the nervous condition, and the local symptoms. As regards the first, there is a general consensus of opinion against the employment of narcotics and alcohol. Such pathological conditions as gout, rheumatism, syphilis, struma, anæmia, and oxaluria must be treated *secundum artem*. The patient should have good food, fresh air, change of scene and climate, warmth, and tonics judiciously selected. Sleep should be procured naturally, and for this purpose exercise, especially in the open air, is advisable.

The nervous condition should be treated by moral means. The will should be educated. There should be no threats or forcing (Jolly). And the patient should be separated from her friends.

Hypnotic suggestion has been tried and has succeeded in some cases. I have seen it signally fail.

Now with regard to the treatment of the local symptoms, seeing that these, in hysterical cases, are imaginary and fictitious, their treatment must necessarily be largely moral and through the nervous system.

Holmes tells us to commence with the removal of all possible irritants. These are not always easily discovered. We must investigate the digestive, nervous, circulatory, and sexual systems.

In regard to strictly local applications opinions vary. Of such simple things as heat and cold, we have Paget, on the one hand, stating that warmth is necessary and cold objectionable, while Jolly holds the converse opinion. Probably the question really is, which would have the greater moral effect in any given case. Galvanism also should be employed according to its moral influence. Counter-irritation is often very useful and is best applied lightly and frequently; moral influence again being desired mainly.

Massage has great possibilities, employed both on the Weir-Mitchell principle and locally to a joint or back. Massage should always be applied and administered under the advice and supervision of a doctor.

Surgical operations are not, in my opinion, advisable. They may be temporarily useful in removing a painful part (such as the mamma or testicle), or by leading the patient to believe that some disease has been removed or relieved (moral influence); but the ultimate result is unsatisfactory. The pain, which has been temporarily relieved, recurs in some other part. In a word, the real disease, the hysteria, is not cured, and cannot be cured by such means. There is another kind of surgical operation which may under exceptional circumstances be desired, viz. when a definite diagnosis cannot be arrived at.

Under these circumstances an exploratory incision may be thought of. Personally I do not approve of such operations. To begin with, they are an admission of failure. It is said that the slight operation can do no harm, and that the moral effect may be very considerable, the patient being led to believe that a curative operation has been performed. As I have already said, the local effect soon passes off, and the patient finds out that she has been deceived, and the moral effect is unfavourable. On the other hand, it is said that if no disease be found the patient will be convinced on being told this. That is not my experience. I have

twice made exploratory incisions (under protest), and in both instances the moral effect on the patient was nil.

If the case be a hysterical one the treatment must be directed to the hysteria. Too much attention may be directed towards the supposed affected part by treatment being applied there. Paget dwells strongly and forcibly on this.

I will, in conclusion, repeat that diagnosis is the main thing. If the case be hysterical, treat the hysteria. If, on the other hand, you have any suspicion that even a trace of disease is present, treat the local affection. It is the less dangerous mistake of the two.

Hystericism.—The hysterical state.

Hystero-cataphraxis.—Fixation of the uterus to the anterior abdominal wall, as in cases of persistent prolapsus; abdominal hysteropexy.

Hysterocele.—A hernia in which the uterus constitutes one of the contents.

Hysterocleisis.—A plastic operation in which the cervical canal is closed; it has been employed in inoperable cases of vesico-uterine fistula.

Hystero-cystocleisis.—A gynæcological operation, performed in cases of uretero-uterine or of vesico-utero-vaginal fistula, in which the cervix uteri is turned into the cavity of the bladder and fixed there.

Hysterodynia.—Pain in the uterus.

Hystero-Epilepsy. *See* Epilepsy (*Diagnosis*); Hysteria; and Hypnotism (*Therapeutic Uses*).

Hysterogenic Points.—Hyperæsthetic areas or zones of skin, pressure upon which leads to the development of an attack of hysteria. *See* Hysteria.

Hysteroma.—A fibroid tumour of the uterus. *See* Uterus, Non-Malignant Tumours of.

Hysterometry.—Measurement of the length of the uterus, as carried out by means of a graduated sound or hysterometer. *See* Gynæcology, Diagnosis in.

Hysteromyomectomy.—The operation by which that part of the uterus which contains a fibroid tumour (myoma) is removed.

Hysteromyomotomy.—Removal of a myoma from the uterus by incision of the latter.

Hysteroneurosis.—A nervous disturbance due to uterine disease.

Hystero-oophorectomy.—The removal of uterus and its annexa, as in Porro's Cæsarean section; Cæsarean hysterectomy.

Hysteropexy.—The fixation of the uterus either to the anterior abdominal wall or to a neighbouring structure or organ, as carried out by the abdominal route (abdominal hysteropexy or ventral fixation) or by the vaginal (vaginal hysteropexy). *See* Uterus, Displacements of the.

Hysterophore.—An instrument for replacing the uterus (in inversion or prolapse) or for maintaining it in its normal position.

Hysteroptosis.—Prolapse of the uterus, or, sometimes, inversion of that organ.

Hysterostomatotomy.—Enlargement of the os uteri by incisions, *e.g.* in forced delivery.

Hysterotomy.—Incision of the uterus.

Hystricismus or **Hystriciasis.**—Ichthyosis Hystrix Gravior (Gr. ὕστριξ, a hedgehog). *See* Ichthyosis.

Hythe. *See* Therapeutics, Health Resorts (*English*).

Iater or **Iatros.**—A physician (Gr. ἰατηρ, a physician or surgeon); many compound words are formed containing this root, such as *iatrochemia* (medical chemistry), *iatrologia* (the science of medicine), *iatrophysics* (physics applied to medicine), and *iatrophylactes* (one fond of taking remedies for the preservation of health).

Ice. *See* Hydropathy (*Use of Ice*).

Ice-cream. *See* Toxicology (*Poisoning by Food-stuffs, Milk, Ice-creams*).

Iceland. *See* Balneology (*Norway, Sweden, and Iceland*); Therapeutics, Health Resorts (*Foreign*).

Iceland Moss. *See* Diet (*Vegetable Foods, Lichens*).

Ichor.—An acrid or irritating discharge from an ulcer or wound.

Ichthalbin.—A compound of ichthyol with albumin (albuminate of ichthyol), given in doses of 15 to 30 grains as an intestinal antiseptic.

Ichthargan.—A silver preparation (*Merck*), being thiohydrocarburosulphonate of silver, soluble in water and glycerine, and recommended in the treatment of gonorrhœa in the male or female.

Ichthyol.—Ammonium sulpho-ichthyolate; ammonium ichthyolsulphonate. A dark-brown viscous substance, with a disagreeable tarry

odour, containing about 15 per cent of sulphur, obtained by the distillation with sulphuric acid and neutralisation with ammonia of a bituminous quartz of fish origin found in the Tyrol. It is soluble in water, glycerine, ether, fats, and oils. *Dose*—10-30 grs. Sulpho-ichthyolates of sodium and lithium may be administered in similar doses. A sulpho-ichthyolate of zinc is also prepared, and is used externally.

Externally ichthyol has been employed in a great number of chronic skin diseases, such as eczema, psoriasis, acne, and favus. It is usually prescribed as an ointment in strengths of from 10-50 per cent. In some cases collodion 7 parts with ichthyol 1 part will be found to be a useful application. It was at one time strongly recommended in the treatment of erysipelas, but it probably has little if any effect in checking this disease. A weak ointment has been said to exercise a favourable influence on the course of small-pox if applied in the early stages. It is occasionally used in the form of an ointment to relieve pain in chronic and subacute articular rheumatism. For gonorrhœa, cystitis, leucorrhœa, etc., injections varying in strength from 1-5 per cent are indicated. Vaginal tampons soaked in ichthyol and glycerine are extensively employed in the treatment of various pelvic inflammatory and congestive conditions. Internally ichthyol is given in capsule or in solution for many skin diseases of a chronic nature, and in urticaria. It is also recommended for chronic rheumatism, and as an antiseptic in flatulent dyspepsia. It has enjoyed a temporary popularity in the treatment of many widely different conditions, but the results obtained have been quite inconclusive in the majority of instances.

Ichthyosis.

Syn.: *Fish-skin Disease, Xerodermia Ichthyoides.*

See also Pellagra (*Symptoms*); Pregnancy, Pathology, Intra-Uterine Diseases (*Fœtal Ichthyosis, etc.*); Skin, Diseases of Sweat and Sebaceous Glands (*Seborrhœa*); Tongue (*Leukokeratosis*).

Definition.—A disease present at birth or developing in early infancy, in which the skin is dry, rough, and scaly, with deficiency of normal secretions; or covered partially or generally with horny plates and warty growths. Under this definition are included all the forms of fœtal ichthyosis, the various degrees of ordinary ichthyosis, and the generalised and localised types of ichthyosis hystrix. "Local ichthyosis" or "hyperkeratosis," due to chronic local irritation, nervous diseases or venous stasis, will also be considered. The following conditions are omitted as not being true forms of ichthyosis, viz. "acquired ichthyosis," a disease of advanced life, and due to a form of seborrhœa. "Ichthyosis palmaris et plantaris" and "ichthyosis linguæ" will be found under the headings of Tylosis and Tongue respectively.

Ichthyosis Fœtalis Gravior—(Syn.: *Harlequin Fœtus*).—In this rare and most repulsive disease changes in the skin of a most pronounced kind are observed at birth. The epidermis is greatly hypertrophied, hardened, and broken up into scales and plates of different sizes, shapes, and thicknesses, separated by fissures and furrows of varying depth and width. The skin feels hard and leathery, and is of a dirty grey or yellow colour. The fissures and furrows, in contrast to the general colour of the plates, are of a deeper purple or brown. As a result of general contraction of the thickened integument, the mouth gapes, and the nose, as well as the reproductive organs, are practically absent. For the same reason the limbs maintain their intra-uterine position of flexion. The mucous membranes are unaffected.

Such children are almost always born prematurely, while owing to the inability to suck, interference to the functions of the skin, suppuration from pyogenic organisms, visceral and pulmonary congestions, death soon ensues. In one case (Jahn) life was prolonged for nine days.

Etiology.—In about 8 per cent of recorded cases other children in the same family have been born with the same disease.

Pathology.—Various views have been expressed as to the minute changes occurring in the skin.

There is little doubt that the onset of the disease dates from the third or fourth month of fœtal life. Some observers (Simpson, R. Croker) hold that the condition is an exaggerated form of ordinary ichthyosis, while others (F. Hebra, Bland-Sutton) consider it to be a form of general seborrhœa.

Unna disputes its relation to ichthyosis, and looks upon the malady as a universal congenital hyperkeratosis due to defective nutrition.

Ichthyosis Fœtalis Mitior.—Under this name are included the milder cases of fœtal ichthyosis.

Family prevalence as in the grave form is not uncommon, and many cases are prematurely born and die young. In a certain number of cases which reach adult life the condition becomes indistinguishable from one of severe ordinary ichthyosis, and for this reason it may be looked upon as a connecting link between ichthyosis fœtalis gravior and ordinary ichthyosis.

At birth the body is covered with a thin dry

pellicle of dark brown or yellowish colour, and after a few days epidermic scales and plates of various sizes are found, with fissures and cracks. The mouth, nose, ears, and genitals are affected as in a harlequin fœtus, but to a less degree.

Pathology.—Caspary found great hypertrophy of all the layers of the epidermis, and a pronounced atrophy of all structural elements of the skin, with the exception of sweat glands, which were in marked evidence.

Ichthyosis Simplex—(Syn.: *I. Vera, Common Ichthyosis*).—Although the more severe forms of this disease were clearly recognised by older observers, Willan was the first to take a comprehensive view of the disease in all its degrees of severity. His original clinical description remains, even at the present day, almost perfect and complete; and his separation of two distinct types—I. simplex and I. cornea—is still generally accepted.

Amongst others Alibert, Rayer, Hebra, Gaskoin, and Erasmus Wilson have greatly helped to advance our knowledge of the disease, while Unna has recently made important observations on its pathological anatomy.

Symptoms.—In the mildest form, to which the names *xerodermia* and *xerosis* are often unnecessarily given, the skin in certain regions, such as the extensor surfaces of the arms and legs, is dry and dirty-looking, with exaggeration of the natural skin lines.

The surface is covered with fine scales, and the hair follicles stand out as innumerable little conical projections, which feel remarkably rough and hard when the hand is passed over them. (*I. anserina vel. keratosis follicularis.*)

Associated with the rough condition of the skin there may be a certain amount of redness, which is liable to become more pronounced in cold weather, or from irritation of any kind. In more severe cases (*I. nitida*) the whole of the surface of the skin is more or less involved, the disease being still more pronounced on the extensor surfaces of the extremities, as in the milder cases. The general appearance of the skin varies with the size, thickness, and colour of the scales.

When the scales are small they desquamate as thin whitish flakes, but when they are large they are of a dirty grey or brown colour, and more adherent. In the latter case the skin is dry, rough, and dirty-looking, the natural lines and furrows are exaggerated, and the skin loses its normal elasticity.

With a further degree of severity the scales are represented by large, thick, polygonal epidermic plates, separated by deep furrows (*I. serpentina, sauridermia*), and adherent to the skin.

The colour in such cases becomes dark brown, green, or even black (*I. nigricans*).

With regard to distribution the disease is most marked on the tips of the elbows, on the fronts of the knees, the extensor surfaces of the extremities, and on the buttocks. The following regions are either affected to a slight extent or are entirely free, viz. the face, where the skin may be rough on the forehead and cheeks, the axillæ, the flexor bends of the elbows and popliteal spaces, the groins, and the genital organs. In nearly all cases the disease is accurately symmetrical in distribution and intensity.

The palms and soles are, in the writer's experience, in most cases hard, dry, and smooth, with exaggeration of the major natural lines and diminution of the minor ones. This opinion is at variance with the statement expressed by Unna and others that the palms and soles are never affected.

The mucous membranes are never involved.

Subjective Symptoms.—Apart from the cutaneous defect subjects of ichthyosis show no other signs of abnormal development and enjoy average good health. Associated with the condition of the skin are certain symptoms, of which a peculiar sensitiveness to changes of temperature, especially to cold, is perhaps the most common. Patients will often volunteer the statement that they "feel the cold terribly."

In many cases there is an entire absence of symptoms, although pruritus, sometimes severe, may give trouble.

Asthma is sometimes associated with the disease, its severity corresponding with the condition of the skin (Jamieson). Acne, boils, and other skin lesions due to pyogenetic organisms are liable to occur, no doubt from the fact that the rough cracked skin is extremely suitable for harbouring micro-organisms.

In addition to these symptoms ichthyotic skin is very liable to be attacked with a moist catarrh or troublesome dermatitis. These inflammatory changes are apt to occur even in the mildest cases from irritation of the skin, exposure to cold winds and heat, or even without any obvious cause.

The recognition of this tendency to inflammation is of the greatest importance both for diagnosis and treatment, for many cases of so-called chronic eczema may be entirely due to this cause, and the underlying condition of ichthyosis may be overlooked.

The persistence of this eczematous condition, starting as it does in early life, may produce a clinical picture closely resembling prurigo. The hair tends to be scanty and lustreless, and is often absent on the trunk and limbs.

The nails are sometimes badly developed, dry, and brittle.

Sweating is diminished in regions where the ichthyosis is pronounced, but may be vicariously increased in parts slightly affected (Aubert). Free sweating in summer gives relief to patients, while wintry weather tends to aggravate any symptoms which may be present. Occasionally,

however, temporary improvement occurs in the winter.

Course.—Ichthyosis is generally observed in the first few months of life, and is not often well marked before the third year is reached.

After this age the disease becomes gradually more marked as the child grows older, and reaches its maximum of severity at about the age of ten years, thereafter persisting throughout life, perhaps improving somewhat in summer, only to relapse again in the winter.

The fact that the disease appears some months after birth, may in part be explained by the action of the amniotic fluid during intra-uterine existence, which prevents the skin from becoming dry; and in addition to this the frequent washing of infants tends to keep the skin soft, and so reduce to a minimum the early evidences of the disease.

When cases are reported as being cured, or as having started after the period of childhood, an error of diagnosis must always be suspected.

Temporary improvement may and often does occur after specific fevers. The disease in all cases must be considered as an incurable one.

Causation.—Heredity appears to be the sole factor in its causation, and ichthyosis may be looked upon as the most hereditary of all skin diseases.

In a large number of cases, relations either in direct or collateral branches of the family are similarly affected.

Although males and females suffer in about equal proportions, there is a remarkable tendency for the disease to be limited to the children of one sex in a family.

As in all other congenital affections, parents will sometimes offer explanations for the appearance of the disease in their children. These are generally of the nature of "maternal impressions."

Ichthyosis is stated to be endemic among two Albanian tribes on the shores of the Adriatic (Tablonowski), and also in Hayti and Paraguay.

Differential Diagnosis.—In ordinary cases there is seldom any difficulty in recognising the nature of the disease. The rough, dirty skin, more marked on the extensor surfaces of the limbs, its onset in infancy, its persistence through life, and family distribution, together form a well-defined clinical picture.

In the first few weeks of life it may be confounded with a condition described by Hallopeau and Watelet as "exfoliation lamelleuse des nouveaux-nés," which is probably identical with the ichthyosis sebacea of Hebra.

In this condition the epitrichial layer of cells, which normally is cast off in the seventh month of fœtal life, persists at birth in the form of a thin, smooth pellicle like a layer of collodion which is gradually cast off for about two weeks, the infant after that period having a perfectly normal skin.

Morbid Anatomy and Pathology.—Microscopical examination shows the condition of the skin to be due to an exaggerated cornification of the cells of the rete Malpighii with very pronounced thickening of the horny layer.

The stratum granulosum, so conspicuous in sections of normal skin, is entirely absent.

The rete Malpighii, according to some observers, is thickened, and its cells abnormally large. Others hold that thinning of this layer is characteristic.

The hair follicles, with the sebaceous glands, are in an atrophic condition, while the sweat glands in most cases appear to be perfectly normal.

The opinion generally held is that the disease is a primary defect of the cornification of epidermic cells, and that consequently it should be looked upon rather as an inherited malformation of the skin than an active morbid process.

Associated with the changes in the epidermis, inflammatory changes are sometimes observed in the true skin, and are considered by most observers to be secondary to the primary disease.

Unna, however, is of an opposite opinion, and concluding that the inflammation is primary, he classifies the disease as an "Infectious inflammation."

Treatment.—The disease, it must be remembered, is practically incurable, but treatment, if carefully carried out, may materially alleviate and keep it under control.

The treatment should be directed with a view to remove the dirty and unsightly scaliness, to keep the skin soft with artificial lubricants, and to protect it from any irritation liable to set up the dermatitis which is so apt to occur. Warm baths, to which are added alkalies, borax, bran, or starch, and the use of alkaline soaps, are very suitable for mild cases and especially for children.

For more severe cases in addition to the daily baths, glycerine, diluted to suit the tolerance of the patient, should be frequently rubbed into the skin. By this means the skin becomes soft and smooth, and may be kept in this condition by regular use of lotions containing glycerine, or by the use of lanolin, vaseline, cold cream, or other fatty substances.

When the epidermis is too thick to be removed by these simple means, salicylic acid in the form of an ointment, cream, or paste will be found useful.

To protect the skin from irritation and changes of temperature, flannel should be worn, and its thickness reduced in warm weather. If itching is troublesome the flannel underclothing may be lined with thin washing silk.

Internal treatment is generally supposed to be of little practical use—Besnier, however, recommends frequent small doses of arsenic, while Brocq highly commends cod-liver oil. The

writer has seen markedly good results from the use of thyroid extract, quite comparable to those obtained by the same drug in myxœdema. Five grain tabloids once or twice a day given after or during meals are the handiest form of administration. The well-known constitutional effects of the drug must, of course, be carefully looked for, and the treatment stopped or suspended on their manifestation.

Ichthyosis Hystrix Gravior.—Syn.: *Porcupine skin, hystricismus, leontiasis hystrix.*

Definition.—A condition arising in early infancy in which the skin is generally, but not universally, covered by localised warty epidermic growths or horny masses.

Historical.—No description of this rare disease is complete without reference to the famous Lambert family of which several members appeared in England, France, and Germany as "porcupine men" at the end of the last and beginning of the present century.

The first case observed in the family was a man in whom the disease appeared when he was about two months old. He had six children, all males, who all developed the disease at the same age. Five of these sons died in childhood, and the remaining one had nine children, two boys and seven girls. The sons again were similarly affected, while the daughters were all unaffected. The disease appeared again in the next generation in a male child of one of these sons.

The disease was therefore present in ten individuals in four generations. In some cases the horny growths were shed at regular intervals.

Clinical.—In the majority of cases the disease appears at the age of about two months, and becomes well marked at an early age.

The general appearance of a case of hystrix may be gathered from the suggestive names given to the condition.

Ichthyosis Hystrix Mitior. — Syn.: *I. hystrix partialis; neuropathic papilloma; nerve nœvus; nœvus verrucosus, linearis, unius lateris.*

Historical.—The earliest cases were recorded by Thomson in 1829 under the name of "papillary nævus."

Bärensprung, in 1863, pointed out the distribution of the lesions and its similarity to the distribution of Herpes Zoster.

Symptoms.—The condition, although probably always present at birth, may not be noticed until the lesions become more pronounced and papillary at the end of two or three months.

At this age the lesions show their characteristic grouping in lines or streaks, and are more or less soft and brownish in colour.

As age advances the lesions tend to become more prominent, harder, and of a deeper colour. They consist of papillomatous or warty growths either arranged in patches or lines and streaks, which tend to run transversely on the trunk and longitudinally on the limbs.

The skin surrounding the lesions is often deeply pigmented. The presence of these warty growths does not affect the general health. In some cases itching is complained of, while in other cases the diseased areas may become inflamed. If the lesions are in such situations as the axillæ, they may become very vascular and grow rapidly. They are then liable to bleed, fungate, and ulcerate with an offensive discharge.

Mucous membranes are as a rule not affected, but Church describes a case in which the palate, tongue, and inside of the cheek were affected, and Ryan saw a case in which the palate was alone involved.

In some cases epilepsy and other nervous manifestations are associated with the disease.

Causes.—The sexes are affected in about equal proportions, and there is no evidence either of family prevalence or hereditary transmission.

The distribution often corresponds to some cutaneous nerve, and sometimes follows the deep nerves of the part. In some cases, as pointed out by Phillippson in 1890, the lines and streaks correspond to "Voigt's lines," *i.e.* the lines of intersection between the areas of distribution of the cutaneous nerves. In other cases the distribution appears to correspond to the lymphatics of the part (Heller), or to metameric developmental fissure-lines (Unna).

Pathological Anatomy.—The papillæ of the corium are markedly enlarged, the connective tissue is loose in structure, the elastic fibres are either atrophied or entirely absent, and the arteries enlarged. The stratum corneum is greatly hypertrophied.

Kerato-hyalin and eleïdine are abundant in the granular layer which is well developed. The rete Malpighii is reduced in thickness and deeply pigmented in its lowest layer.

Dilatation of the sweat ducts is a marked feature in many cases, but the sebaceous glands and ducts are normal.

Treatment is generally only necessary when the lesions are unsightly from being situated on the face, neck, or hands, or when the local irritation or inflammation gives much trouble.

For small growths temporary or even permanent relief may be given by using a strong solution of salicylic acid or resorcin in collodion, or by repeated paintings with tincture of iodine.

For larger growths, free removal either with the galvano-cautery or with the knife is to be recommended.

Local Hyperkeratosis.—Under this name are considered several conditions in which thickening of the horny layer of the skin occurs in localised patches.

This thickening may be found on any part of the body, but is more commonly seen on the palms of the hands and soles of the feet, a fact no doubt explained by the normal thickness of the epidermis and the liability of these parts to frequent and various forms of irritation.

Callositas.—The most common form of simple hyperplasia of the horny layer is seen in the callosities, which may develop on any part of the body subjected to continuous pressure or irritation.

These callosities frequently occur in the hands of workers in various trades, such as mechanics, shoemakers, carpenters, tailors, and others, and are directly caused by the irritation resulting from the continued use of hand tools. Similar changes occur on the finger tips of stringed instrument players, on the hands of oarsmen, and the hands of metal workers who handle mineral acids.

The callosities vary in position according to the particular kind of tool used, and, as Hebra pointed out, the occupation of a man may often for this reason be ascertained by examination of the hand.

It must not be forgotten, however, that callosities may develop in other regions, such as under badly fitting belts or trusses, and on the feet from ill-fitting boots.

Local Hyperkeratosis associated with general Skin Diseases.—When the palms and soles are attacked in such diseases as eczema, psoriasis, lichen planus, and syphilis, the lesions often occur as localised patches with marked thickening of the horny layer. This hyperkeratosis tends to mask the usual characters of such eruptions in these regions, and in the absence of lesions on other parts of the body it may be difficult to make a positive diagnosis of the condition.

Of these conditions the most pronounced hyperkeratosis is found in the tertiary syphilide localised on the sole of the foot.

Arsenical Keratosis.—This condition is caused by the long-continued use of arsenic, and is usually accompanied by arsenical pigmentation, peripheral neuritis, or other manifestations of chronic arsenical intoxication.

The eruption which generally appears first on the hands, and more rarely on the feet, consists primarily of patches of small warty growths situated round the orifices of the sweat ducts, and subsequently of callosities with a roughened dry condition of the intervening skin. Chronic hyperidrosis is a frequent concomitant, and it may precede the development of the hyperkeratosis by a considerable period. As the lesions enlarge they tend to lose their nodular appearance and become diffused, raised, thickened, rough patches. These changes tend to persist indefinitely even after discontinuance of the drug.

The most important point in connection with arsenical keratosis is its liability to become the seat of epithelioma.

Other forms of Local Hyperkeratosis.—Prolonged hyperidrosis of the palms and soles, common in neurotics, excessive tea drinkers and alcoholics, may lead to considerable horny thickening, the skin becoming smooth with loss of the smaller natural lines. Occasionally considerable pain and tenderness is complained of, and painful cracks and fissures may form.

Local horny thickenings have been observed as a result of venous stasis as a sequel to various nerve injuries, and during the course of tabes dorsalis.

Of the more rare forms of hyperkeratosis may be mentioned the *keratodermia erythematosa symmetrica* of Besnier, in which there arises in early life multiple scaly patches on the palms and soles surrounded by a narrow erythematous zone.

The condition is not hereditary like tylosis, and its cause is unknown. It gives rise to no severe symptoms, and persists throughout life.

A somewhat similar affection has been described by Brooke under the name of *erythema keratodes symmetricum.*

A sharply defined erythema appears on the palms and soles, and is followed by a superficial hyperkeratosis with some œdema and tenderness. This condition is readily cured, and appears to be more of the nature of a primary inflammation.

Treatment.—When the epidermic thickening is associated with some general disease the treatment should be modified accordingly.

In cases where the condition is due to chronic irritation or continuous pressure these causes must be removed if cure is to be permanent.

For local treatment the thickened tissue should be softened and pared down with a knife.

For softening the surface the skin may be bathed with hot water and soft soap, and afterwards treated with wet dressings, such as solutions of caustic potash, salicylic acid, or acetic acid. The plaster muslins made by Beiersdorff, of Hamburg, containing salicylic acid, creasote, resorcin, and pyrogallol, if persistently used, may bring about a permanent cure.

Ichthyotoxin. *See* Snake-Bites and Poisonous Fishes (*Eels*).

Ichthysmus.—Poisoning by the eating of fish.

Icterus.—Jaundice. *See* Contusions (*Clinical Features, Traumatic Icterus*); Hæmatemesis (*Causes and Source*); Heart, Affections of Myocardium and Endocardium (*Symptomatology, Enlargement of the Liver*); Liver, Diseases of (*Chronic Venous Congestion of the Liver*); Liver, Diseases of (*Hepatic Tuberculosis*); Liver, Diseases of (*Icterus Gravis, Acute Yellow Atrophy*); New-Born Infant (*Icterus Neonatorum*); Umbilicus, Diseases of (*Hæmorrhage, Sepsis*).

Ictus Epilepticus.—An epileptic fit. *See* Epilepsy.

Ictus Solis.—Sunstroke (*q.v.*).

Idea.—A concept or "mental representation of an object of sense." *Chase-ideas* are those which pass rapidly through the brain, which are easily produced, and which make but a slight impression. *Fixed-idea* is a form of monomania, in which the patient's conduct and thoughts are all under the influence of one idea.

Ideational Insanity.—A form of insanity characterised specially by perversion of the reasoning powers.

Identification. *See* ANTHROPOMETRY; MEDICINE, FORENSIC (*Identity*).

Idioctonia.—Suicide (Gr. ἴδιος, personal, and κτόνος, murder).

Idiocy. *See* MENTAL DEFICIENCY. *See also* ADOLESCENT INSANITY (*Significance*); BRAIN, ATROPHY, HYPERTROPHY, CYSTS, ETC.; CHILDREN, DEVELOPMENT OF (*Development of Special Senses, Hearing, etc.*); DEAFMUTISM; HYDROCEPHALUS (*Symptoms*); PARALYSIS (*Cerebral Diplegia*).

Idiopathic.—A primary morbid state is called *idiopathic* when it is regarded as arising by itself in the part affected; it is opposed to a *symptomatic* malady or one dependent upon another disease elsewhere. *See* ADRENAL GLANDS, ADDISON'S DISEASE; MUSCLES, DISEASES OF (*Idiopathic Muscular Atrophy*); PERITONEUM, ACUTE PERITONITIS (*Idiopathic or Cryptogenic*).

Idiosyncrasy. A peculiarity, especially a susceptibility to be markedly influenced by certain morbid agencies or medicinal preparations.

Idiot and **Idiotism.** *See* IDIOCY; MENTAL DEFICIENCY.

Ignipuncture.—Puncturing a part or organ of the body (*e.g.* the cervix uteri) with the cautery.

Ikao. *See* BALNEOLOGY (*Japan*).

Ikaria. *See* BALNEOLOGY (*Greece*).

Ikota.—A hysterical condition or form of religious insanity occurring in Siberia.

Ileo-.—In compound words *ileo-* means relating to the ileum or the third part of the small intestine.

Ileo-cæcal Region. *See* APPENDICITIS (*Anatomy*); TYPHOID FEVER (*Symptoms*).

Ileo-colitis.—Inflammation (enteritis) of ileum and colon. *See* GASTRO-INTESTINAL DISORDERS OF INFANCY (*Inflammatory Diarrhœa*).

Ileo-colostomy.—The making (by operation) of an artifical communication between the ileum and the colon.

Ileo-ileostomy.—The making (by operation) of an artificial communication between two parts of the ileum.

Ileus. *See* INTESTINES, SURGICAL AFFECTIONS (*Intestinal Obstruction*).

Ilfracombe. *See* THERAPEUTICS, HEALTH RESORTS (*English, Devon*).

Iliac.—Relating to the ilium, *e.g.* iliac fossa, iliac region, etc.

Iliacus Muscle. *See* SPINE, SURGICAL AFFECTIONS OF (*Neuroses of Lumbar Plexus*).

Ilio-.—In compound words *ilio-* means relating to the iliac bone, *e.g.* ilio-femoral, ilio-inguinal, ilio-pectineal, ilio-trochanteric, etc.

Ilkley. *See* THERAPEUTICS, HEALTH RESORTS (*English, Yorkshire*).

Illegitimacy.—In vital statistics the proportion of illegitimate births has a marked influence upon infantile mortality; where the illegitimate births are numerous the infantile deaths are greatly increased; in this country it is stated that the fall in the illegitimate birth-rate of recent years is greater than that of the legitimate birth-rate.

Illumination Test. *See* NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Diagnosis, Illumination Test*).

Illusion.—The "false interpretation of a sensation actually perceived," or "erroneous mental conception of an external object perceived by one of the senses." *See* ALCOHOLISM (*Chronic, Clinical Features*); PHYSIOLOGY, SENSES (*Vision, Optical Illusions*).

Imbecility. *See* MENTAL DEFICIENCY. *See also* ALCOHOLIC INSANITY (*Dementia*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Results*); BRAIN, INFLAMMATIONS (*Acute Encephalitis, Clinical Features, Sequelæ*); CRETINISM; HEAD (*Shape in Mongolian Imbecility*).

Imitation. *See* CHOREA (*Etiology*).

Immigration.—In vital statistics much immigration favours an actual increase of population; and when conjoined with the natural increase (as shown by a high birth-rate and a low death-rate) will greatly favour it (*e.g.* in the United States of America). *See* VITAL STATISTICS (*Population*).

Immorality. *See* INSANITY, NATURE AND SYMPTOMS (*Insane Diathesis, Degeneracy*); VICE (*Medical Aspects of*).

Immune. *See* Immunity.

Immunity.

See also Anthrax; Blackwater Fever (*Epidemiology*); Epidemiology (*Immunity*); Plague; Rabies (*Immunisation*); Therapeutics, Serum Therapy; Undulant Fever (*Etiology, Immunity*); Vaccination; Yellow Fever.

Introduction.—The conception of immunity was first formed when physicians learnt that after recovery from serious diseases, and especially after small-pox, a life-long protection may be secured from like diseases. Jenner then pointed out that enduring protection against a disease can be acquired by undergoing a similar or nearly related disease (as in vaccinia and small-pox), and since 1880 Pasteur's discoveries in regard to various artificial protective inoculations have given us a clearer conception of the nature of immunity. Gradually thereafter arose the questions, which are of both theoretical and practical interest, as to why man is not susceptible to so many parasitic diseases, and as to how the living organism is enabled to withstand the putrefactive bacteria, whether they be those of the intestinal tract or of the external surface of the body, which manifest their destructive power immediately after the death of the whole body or of individual parts. In both these instances, however, we are obviously dealing with other circumstances and conditions than in the case of protective inoculations. It is customary, therefore, to differentiate "natural" or "innate immunity" from "acquired" or "artificially induced immunity." But in order that the subject may be clearly understood it is important that our nomenclature should indicate the existence of this fundamental difference, hence in the following pages the innate condition of immunity will be designated as "natural power of resistance," while the term *Immunity* will be reserved exclusively for the specific condition which has either been acquired or has been artificially produced by protective inoculation. In short, specific immunity, on the one hand, is characterised by the presence in the body of specific antitoxins, or, speaking generally, of specific anti-substances which have been formed in the body in consequence of undergoing a specific disease, or by means of specific treatment. On the other hand, the natural power of resistance depends chiefly on the capability of the leucocytes to act either as phagocytes or as elaborators of microbicidal alexins, which are totally different in constitution and mode of action from antitoxins and anti-substances. Again, it

should be noted that there are two main types of infective processes: one in which the morbid changes are caused by great multiplication of the virus in certain parts of the body or throughout the blood generally, for example, anthrax, and a second type in which the virus remains localised at the seat of invasion whilst the morbid changes of the body in general are due to the absorption thence of toxic products formed locally, for example, tetanus. We can thus differentiate "pure bacterial infection" from "toxic bacterial infection," and it is therefore only natural, when dealing with specific immunity, that "simple immunity to bacteria" and "immunity to toxin" or "tolerance of poison" should be distinguished from one another. It is obvious that we cannot always draw such hard and fast lines, and many infective processes represent an admixture of the two types. We may regard anthrax, tuberculosis, leprosy, and glanders (malleus) as examples of "pure bacterial infection,"—tetanus, diphtheria, and Asiatic cholera as instances of "toxic infection,"—typhoid fever, plague, influenza, septico-pyæmia, etc., as being "mixed forms." From a practical standpoint immunity to toxin is of more importance than immunity to bacteria, because in toxin infections, for example, diphtheria or tetanus, the transference of antitoxic serum from a specifically treated—actively immunised—animal may confer very marked protection, and may induce a cure if there has been pre-existing infection (*passive* immunisation), whilst in pure bacterial infections such protection cannot be conferred by the blood serum of an actively immunised animal, for instance in anthrax, tuberculosis, etc. In the mixed forms the success attained by passive immunisation, *i.e.* by serum therapeutics, is varied, but usually slight. By active immunisation, *i.e.* by inoculation with killed cultures, however, man may obtain a certain degree of protection from Asiatic cholera and plague (Haffkine).

Another point of practical significance is that the natural power of resistance may be increased by certain means, and that this increase is manifested either throughout the whole body or merely in certain localised parts. In endeavours towards this end medicine touches upon what may be called the question of personal or individual hygiene, which likewise aims at increasing the power of natural resistance against disease.

I. Natural Power of Resistance (Innate Immunity)

A. Natural Resistance to Bacteria.—Each infective agent endangers the various species, varieties, and even races of animals to a different degree, and no infective agent is of the same danger to all. Infective agents have only a limited sphere of action in the animal kindgom, whilst most species, etc., possess a natural power of resistance against them. The human subject, for example, appears insusceptible to cattle plague, fowl cholera, swine erysipelas, etc., whilst all animals are resistant to the micrococcus of gonorrhœa, to scarlatina, measles, influenza, and so on. Even closely allied varieties may manifest great differences in susceptibility; for example, the field-mouse (*Arvicola arvalis*) is very susceptible to glanders and tuberculosis, whilst the common house mouse and white mouse are resistant. The marmot (*Spermophilus guttatus*), which is indigenous to southern Russia, possesses a remarkable susceptibility to glanders. Even racial differences may play an important part. Algerian sheep, for example, are much less susceptible to anthrax than are the races of European sheep. Similarly, certain breeds of swine (Yorkshire hogs) are decidedly more resistant to swine erysipelas than are other breeds. Differences are also found among the races of men. Negroes are noted for their remarkable powers of resistance to yellow fever, and in a lesser degree to malaria, yet they quickly sicken of and succumb to tuberculosis and small-pox.

In identical species and races, age, the state of nutrition, and other circumstances are important factors. Young individuals are in general less resistant. Young pigeons, for instance, may be infected with anthrax, whilst the older birds can be infected only when their physical state has been altered by prolonged fasting. The same holds true for anthrax in dogs, their power of resistance being reduced if water is withheld from them. Tesser and K. Müller by feeding rats exclusively on a bread diet succeeded in rendering them more susceptible to anthrax than when they are kept on a meat diet, and Charrin and Roger obtained similar results by fatigue induced by making rats run in a revolving wheel. Pasteur's experiments on fowls and pigeons are celebrated. The birds' normal temperature (42° C.) was lowered by immersion in cool water, and the power of resistance against anthrax was thus abolished. Many animal experiments undertaken by Wagner, Lode, and others prove that the natural power of resistance to various infections can be lowered by artificial cooling. The injection of chloral, antipyrin, or phloridzin also diminishes the power of resistance. In the human subject, besides such factors, for example, cold, as already mentioned, conditions of psychical depression exert an important influence in lowering the power of resistance, as in tuberculosis. Again, the power of resistance which is commonly present may be suspended by local injury to certain organs, or by local factors which from the outset favour the multiplication of the infective agent, or its transference into the blood. Severe bruising of the periosteum, for example, not infrequently induces pyæmia, in consequence of the staphylococcus settling

down in the contused periosteum. Boils on the lips are known to be very dangerous, because the staphylococci gain access to the blood with unusual rapidity from the tissue of the cheeks, which are rich in lymphatics.

B. Causes of Natural Resistance to Bacteria

We must, in the first place, mention the *external* protective appliances which retard the penetration of infective agents into the body. Provided there be no separation of continuity the external skin affords protection, which is, however, insufficient on inunction of the infective agent with mechanical pressure. There is a second line of defence in the lymphatic glands, which mechanically retain the infective agents taken from the lymphatic vessels, and the organism is thus afforded time to rid itself of the virus by its further protective powers (*vide infra*). Intact mucous membranes are, for the most part, impervious to infective agents. Large quantities of virulent anthrax spores, for example, are required to infect guinea-pigs or mice by the intestinal canal, whilst very small quantities suffice to infect either subcutaneously or by the lungs if the infective material be finely pulverised. Other truly protective appliances of the body are the acid reaction of the normal stomach contents, the bactericidal properties of the saliva, of the vaginal and urinary secretions, and so on.

It is evident that the essential cause of natural resistance to bacteria does not consist in these external protective appliances, but in internal and somewhat complex conditions of the bodily organism of which we have as yet only an incomplete knowledge; yet the facts which we do know are from the practical aspect those of the greatest importance. It was formerly thought that infective agents could not multiply at all in a resistant organism on account of unsuitable food-supply. Such a conception is, as a rule, incorrect, for the bacteria usually multiply at first—for instance, in superficial wounds—yet after a time the multiplication ceases and recovery ensues. Hence the problem is as follows:—What are the factors which effect this change? by what means is the multiplication of bacteria inhibited whilst there is manifestly suitable pabulum for them in the tissues? The change can only be induced by the direct action on the bacterium of the prophylactic appliances of the body two of which are known, viz. phagocytes and alexins.

1. *Phagocytes*, according to Metschnikoff (phagocytic theory since 1884), are in part wandering and in part fixed tissue cells, which, by throwing out protoplasmic processes, are capable of developing, of then digesting, and of thus removing infective agents which have penetrated into the body. The chief wandering phagocytes are the mono- and polymorphonuclear leucocytes (with the exception of the small lymphocytes and of Ehrlich's mast-cells), and the so-called wandering tissue cells; whereas many endothelial cells, especially Kupfer's star cells, the cells of the splenic pulp and of bone-marrow, sometimes also connective-tissue cells, and even nerve and muscle cells, act as fixed phagocytes.

The first decisive observations were made by Metschnikoff in the fungus disease of the daphnia, a species of water-flea, where the fungus spores are devoured by the leucocytes of the daphnia, and a cure thereby effected. Metschnikoff afterwards detected a similar process in frogs affected with anthrax, and more especially in them did he observe the death of anthrax bacilli inside the leucocytes of the frog, a process associated with swelling of and degenerative changes in the bacilli. By numerous observations Metschnikoff later on proved that phagocytosis may be perceived in the course of all infective processes, and more especially if the animal be resistant, *i.e.* if the process end in recovery. If micro-organisms are brought into direct contact with the phagocytes of a resistant organism—for example, if injected into the blood-vessels—they are then taken up by wandering or fixed phagocytes. Should the micro-organisms reach a part where there are few or no phagocytes, the subcutaneous tissue for instance, numerous leucocytes migrate towards the affected spot, and in vertebrates this process is facilitated by the inflammatory emigration. Thus the local deposition of bacteria induces a local reaction which indicates the inflammatory process. The cause of this immigration consists, as proved by Leber, Massart, Bordet, and Buchner, in chemiotaxis of leucocytes, *i.e.* in the capability of their migrations being affected by the attractive influences of certain chemical substances. We make a distinction between positive chemiotaxis (attraction) and negative chemiotaxis (repulsion). Buchner showed that dead bacteria, the proteid constituents of bacterial cells (bacterial proteins), and, lastly, certain closely allied chemical substances, such as vegetable casein, are positively chemiotactic or attractive. The toxins of many virulent bacteria have a negative chemiotactic or repellent power, so that they are avoided by the phagocytes. Bacterial spores may also be engulfed by phagocytes, and even though not killed will yet be prevented from germinating. Metschnikoff has also proved that not only living but also virulent infective agents may be engulfed by phagocytes. Yet, when infective agents which have been enclosed in collodion sacs, and thus protected from phagocytes, are introduced into the tissues, they retain their vitality and virulence a long time, even though the animal be resistant. In natural resistance to bacteria phagocytosis is, according to Metschnikoff, developed to an exceptional degree, and is of such constant and regular occurrence that we

may often foretell from the degree of phagocytosis whether or no the animal experimented on will gain the victory over the micro-organisms.

2. Although the observations of Metschnikoff are undoubtedly correct, yet the "cellular theory," *i.e.* the phagocytic theory, cannot be wholly and unreservedly accepted, because the fluids of the animal body contain not only phagocytes, but also bactericidal substances in a soluble form, which are termed alexins, and which are likewise concerned with the destruction of bacteria. Von Fodor was the first to show that freshly-drawn rabbit's blood can destroy anthrax bacteria—a fact confirmed by Nuttall and Flügge—and he also proved that not only defibrinated blood, but also pericardial fluid and aqueous humour of dogs and rabbits, is bactericidal, and that the blood loses its activity on being heated to 55° C. Buchner, in conjunction with Fr. Voit, Sittmann, and Orthenberger, found that completely cell-free blood plasma or serum has a bactericidal action which is lost at 55° C., but is not altered by freezing and subsequent thawing. Fresh blood, however, when frozen and afterwards thawed loses its bactericidal power, not because the active substances are destroyed, but because the red blood corpuscles are dissolved, whereby the blood becomes a medium so suitable for bacteria that its bactericidal action is compensated for, just as weak antiseptics are if bacteria be well nourished. The destruction of bacteria in serum, however, cannot be referred to lack of nutriment, since serum acts as a nutrient medium after being heated to 55° C. Behring, working with rats which are moderately resistant to anthrax, had demonstrated the destructive action of their blood on anthrax bacilli, and believed that it was to be ascribed to an organic base. Buchner was the first to prove that the protective substances contained in the serum (alexins) are of proteid nature and very unstable.

The proteid nature of these substances is indicated by the proportion of salts contained in the serum. Withdrawal of the mineral salts from the serum by dialysis suspends its bactericidal activity, which is restored, however, by readdition of the salts. But the bactericidal power is not due to the salts alone, for in that case it would be maintained after serum had been heated to 55° C. The action of the salts is consequently an indirect one; it is only when they have entered into loose combination with the proteid alexins that the functional power of the latter can be displayed, just as the functions of the cells and organs of the general body are dependent on their containing a normal amount of mineral salts. Moreover, the alexins can be precipitated along with the other proteid substances of the serum, can be dried and again dissolved without losing their activity. Yet it has hitherto been impossible to truly isolate the alexins because of their instability. They retain their activity for a considerable time in frozen serum, but lose it sooner or later at higher temperature or under the influence of light and oxygen. The alexins of different animal species have different degrees of activity, those of the human serum are very actively bactericidal. The degree of bactericidal activity, however, is largely dependent on the nature of the bacterium employed, and on the relative proportion of serum to the number of bacteria contained therein. A given quantity of serum can only destroy a certain number of bacteria, for the alexins themselves are destroyed or used up by contact with bacteria. This is the explanation of the increased danger to which the body is exposed when the infective agents are numerous. It is almost immaterial whether we inoculate a dead sterile nutrient medium with a few or with milliards of germs of one kind; in both instances they multiply until the nutrient medium is all consumed. In the living body, however, the results will be very different, for resistance is more likely to be maintained and recovery to occur if only a few germs be employed. The action of alexins on bacteria is probably a proteolytic one on the bacterial cell plasma. We may therefore assume that the alexins are deleterious to other cells foreign to the body, *e.g.* they dissolve the hæmoglobin out of the red blood corpuscles of a different animal species (hæmolytic action, *vide infra*). An explanation of natural resistance to bacteria by such action of alexins as just described would constitute a "humoral theory" in contradistinction to the cellular or phagocytic one. But the "humoral theory," too, cannot be accepted *in toto*. The real explanation of the problem lies midway between these two antagonistic theories, for, according to all recent investigation, the alexins are mainly derived from the leucocytes.

3. The leucocytes, as is claimed in the cellular theory, must be regarded as the chief cause of natural resistance to bacteria, but not merely because they are phagocytes devouring and digesting bacteria, but also because they produce alexins. The great bactericidal power of exudations containing leucocytes is due to alexins. It was first proved that the bactericidal action of blood and artificial exudations was proportionate to their richness in leucocytes (Denys, van de Velde, Havet, and Kaisin). But the notion still prevailed that the leucocytes acted as phagocytes, until Hankin and Kanthack indicated that the alexins were a direct product of the eosinophile leucocytes. The experimental proof thereof was afterwards afforded by Buchner and Hahn as follows:—

Exudations which are rich in leucocytes, yet free of bacteria, are obtained by the injection of sterilised emulsion of wheat-gluten into the pleural cavities of rabbits and dogs, and such

an exudation is more markedly bactericidal than the blood and serum of the same animal. This increase of bactericidal power does not depend on phagocytosis, for it is maintained after freezing and then thawing the exudation—a procedure which kills the leucocytes of a warm-blooded animal, but does not destroy the alexins. This was afterwards confirmed by Schattenfroh and Jacob; and Bail and Bordet, working in Metschnikoff's laboratory, proved by a series of experiments that bactericidal substances are supplied to the serum by the leucocytes. Metschnikoff accordingly admits that alexins may be produced by leucocytes, but conceives that they only pass into the serum on the death of the phagocytes ("Phagolysis"), such as occurs in abundance after the withdrawal of blood or after its injection into serous cavities. He says there are no alexins in the normal tissues and the blood within the body because living leucocytes, in his opinion, do not secrete any alexins. Buchner, Laschtschenko, and others dispute this assertion. Whether the question be finally decided in favour of one or other view, yet in either case we would have to conclude that, at the commencement of every infective process, as soon as the normal conditions are altered by the presence of bacteria, phagolysis occurs, *i.e.* death of the phagocytes, whereby alexins get into the locally exuded fluid. But when once there, they must manifest their bactericidal action, and the primary injury to the vitality of the micro-organisms is always therefore due to alexins. This injury is not detectable microscopically, but in consequence thereof the secondary process which may be seen under the microscope, *i.e.* the devouring of the bacteria by phagocytes, is rendered much easier. The detection of virulent bacteria in the interior of phagocytes is no proof against a primary antecedent injury induced by alexins, for the injury may possibly consist merely in a momentary suspension of the vital chemical activity of the micro-organism—a condition of latency, as in bacteria kept at a temperature below 0° C. for example—whereby the elaboration of toxin, and therefore negative chemiotaxis, is momentarily suppressed, whilst positive chemiotaxis due to excretion from the bacterial cell of traces of its proteid constituents alone continues in force. Should the phagocytes afterwards succumb to any cause, *e.g.* to one experimentally induced, the engulfed bacteria may then renew their vital activity, may multiply, and show undiminished virulence.

A view similar to this had formerly been advanced by Bouchard, and such a theory is, it now appears, the most correct. In contradistinction to the phagocytic and humoral theories it may be termed the "alexocyte theory," for the cells by means of their alexins effect partly an intra- and partly an extracellular destruction of bacteria which have invaded the body. Alexins, as products of leucocytes, appear to be related to those histolytic enzymes of the latter (Leber), which cause tissue softening in abscesses, and especially in cold abscesses. The proteolytic action of leucocytes can be experimentally demonstrated by the introduction of little cubes of coagulated egg albumin into the subcutaneous tissue of rabbits; the albumin after a time is seen to be softened and digested. The absorption of catgut in completely aseptic wounds is another instance of the proteolytic action of leucocytes, an action which is in keeping with their fundamental character as absorptive cells. Chemiotactic attraction of leucocytes is not only effected by bacteria and bacterial products, but also by vegetable casein, and by all abnormal animal albumins (Buchner). The leucocytes are therefore not merely combative, but also absorptive cells. On this account the alexins, which must be regarded as proteolytic enzymes derived from leucocytes, cannot be considered as formed for the purpose of affording protection against bacteria, but must be regarded as an appliance common to and indispensable for every animal organism. They normally effect the dissolution of organised substances, yet their action may also be directed against substances and cells which may have penetrated as foreign bodies, whether they be the red blood corpuscles of a foreign species, bacteria, or other microscopic fungi.

C. Increase of Natural Resistance to Bacteria

This may occur in two forms, either generalised throughout the whole organism or locally in particular areas or organs.

1. General increase of natural resistance to bacteria is effected by such agents as induce general hyperleucocytosis, for if the blood contains an excess of leucocytes it possesses increased bactericidal power, unless the leucocytosis be a pathological one, as, for example, in leucocythæmia. Hyperleucocytosis can be effected by various means, but their action is always that of increasing the preventive powers of the body. The following preparations have been hitherto successfully employed subcutaneously:—deutero-albumose prepared by digestion (Matthes and Krehl), organic extracts, spermin, and substances allied to albumose (Goldscheider and Jakob), yeast nuclein and nucleic acid obtained from yeast (Vaughan, M. Hahn), papayotin (Pawlowsky), emulsin, diastase (Hildebrandt), and pilocarpin (Loewy and Richter). Success may also be attained in the human subject (M. Hahn), and according to the results recorded in America the above-mentioned products obtained from yeasts are those whose administration is likely to be the most successful.

Similar effects can be obtained by the use of bacterial proteins—substances extracted from

the cells of various forms of bacteria, because they likewise induce hyperleucocytosis (Buchner). The older tuberculin of R. Koch (1891) belongs to this class of substances, and is to be regarded as essentially the protein of the tubercle bacillus. The specific action which Koch supposed this tuberculin to possess is a very limited one. It induces hyperleucocytosis, however, and acts as an irritant on all the tissues of the body, but by careful dosage the irritation amounts to real inflammation with accumulation of leucocytes only in diseased parts where there is some pre-existing irritation.

As bacterial protein is contained in the bacterial cells, it is only natural that hyperleucocytosis and increase of resistance should be induced by the mere use of killed cultures of various bacteria. It was experimentally shown that subcutaneous injection of killed cultures of the pneumo-bacillus may effect a cure from anthrax (Buchner), and that in typhoid fever subcutaneous injection of sterilised cultures of the typhoid bacillus or B. pyocyaneus may sometimes be beneficial, and may considerably shorten the course of the fever (E. Fränkel, Rumpf). Living bacteria of various kinds may consequently be introduced into the animal body; they perish there, and excreting protein they induce hyperleucocytosis and increase the resisting power. Thus rabbits, after simultaneous infection with anthrax bacilli and non-lethal streptococci of erysipelas, recover from anthrax (Emmerich). A similar recovery of rabbits from anthrax can be effected by means of B. pyocyaneus (Bouchard) or various other bacteria (Pawlowsky). For the same reason a severe intercurrent yet not fatal attack of erysipelas may induce recovery from pre-existing pulmonary tuberculosis (Schäfer, Waibel, Macnamara, Thornton, etc.).

The injection of various apparently absolutely indifferent fluids, for example, physiological saline solution, bouillon, etc., into the peritoneal cavity of guinea-pigs may be followed by a temporary increase of natural resistance, which may even simulate a specific immunisation. Cinnamic acid is an agent exciting general hyperleucocytosis. Landerer has for several years successfully treated pulmonary tuberculosis in man by the use of intravenous emulsions of cinnamic acid. The fine particles of cinnamic acid circulating in the blood are deposited in those parts of the body where there are morbid tissue changes, hence in the tissue around tubercular foci. The results of the chronic inflammation in this instance are dilatation of capillaries, œdema and accumulation of leucocytes; at a later stage a thick wall of leucocytes is formed, there is subsequent formation of connective tissue, and lastly cicatrisation.

Finally, we may note that a general increase of the natural power of resistance to bacteria may undoubtedly be affected by general dietetic and hygienic measures, and the most convincing proof of this is afforded by the successful dietetic treatment of pulmonary tuberculosis at the present day. In this instance nutriment suitable both in quality and quantity, suitable alternation in the use of the different organs, bodily activity, pure air, and all strengthening measures are of great importance. We may note more particularly that general hydrotherapeutic measures which attract great numbers of the leucocytes towards the skin may undoubtedly be followed by general hyperleucocytosis. The same is obviously true in the case of sun and light baths, for there is no doubt that they too attract leucocytes towards the skin.

2. *Local* increase of natural resistance to bacteria is most easily effected by the artificial increase of blood-supply to the part in question. The resistant power of a part may possibly be increased because it is better nourished by blood. Yet the blood must not be regarded solely as a means of transport for nutriment, for it has also, in proportion to the leucocytes and alexins it contains, an important absorptive action on diseased structures, and also, of course, against their causal agents. The experience gained by increasing the blood-supply to diseased parts of the body, such as the limbs, entirely corroborates this view. The most important results in this respect have been obtained by Bier, who was the pioneer of the successful treatment of tuberculosis of joints and bones by means of chronic venous congestion. The following methods of inducing increased blood-supply have been employed:—

(*a*) Venous congestion of a limb by means of elastic compression. This method may be beneficial not only in tuberculosis, but also in gonorrhœal infection of joints, and in acute and chronic articular rheumatism.

(*b*) Arterial hyperæmia is best induced by hot air (100°-150° C. with a special apparatus); for articular rheumatism and arthritis deformans.

(*c*) Mixed hyperæmia induced by Bier's suction apparatus; for chronic articular rheumatism.

(*d*) Increased arterial flow without evident external hyperæmia is caused by the permanent application of alcoholic bandages.

Such bandages were first employed by Salzwedel in cellulitis, lymphangitis, whitlows, boils, mastitis, etc., and with splendid results. In many cases an excellent recovery from tuberculosis affecting bones or joints may follow the permanent use of alcoholic bandages. This action cannot be due, as was supposed, to any direct disinfection by means of the alcohol, for alcohol cannot penetrate deeply into the tissues, and moreover the presence in the tissues of any direct chemical disinfectant is well known to be favourable to bacteria, which, because of their protective membranes, are always more resistant to antiseptics than are the tissue elements.

Alcohol, however, when locally applied has, more than any other chemical agent, the direct power of dilating blood-vessels and especially the arteries. In limbs which are enveloped in alcoholic bandages the effect is seen in local increase of arterial pressure, proving that the action of the alcohol is distributed throughout the whole limb. This is the explanation of the beneficial change which occurs so rapidly in cellulitis, for example, after the application of alcoholic bandages.[1]

D. Natural Resistance to Poison

A certain degree of knowledge concerning innate immunity to poison existed even in ancient times. Lucanus, in the Ninth Ode of *De Bello civili*, mentions that the Psylli, an African tribe, were able to withstand the bite of the asp found in their country. They also understood how to cure strangers bitten by snakes, the treatment mainly consisting in sucking the wound and rubbing in abundance of saliva. It is therefore interesting to note that, according to Wehrmann (*Annal. de l'Institut Pasteur*, 1898), ptyalin has a definite antitoxic action against snake poison.

Ages ago it was known that snake venom is comparatively harmless when in the stomach, and such a "regional" resistance to poison (Behring) is found to be very common; rabbits, for example, are resistant to morphia when administered subcutaneously, intravenously, or by the stomach, but not so to intra-cerebral injections (Roux and Borrel). Similarly with tetanus toxin in the case of fowls; they exhibit considerable resistance to this deadly toxin if it be administered by the usual subcutaneous method, yet are killed, even by small doses, if the injection be intra-cerebral.

Those animals, nevertheless, which are insusceptible to a toxin administered by the usual methods, *i.e.* subcutaneously or intravenously, must be denoted as "resistant to the poison." Now there are differences in the degree of resistance, and two different animal species almost invariably exhibit a different degree of susceptibility to a poison. Knorr, for example, ascertained that the following quantities of the same tetanus toxin are required to produce a lethal effect for each gramme of body-weight:—In the horse 1 unit of toxin, in the guinea-pig 2 units, in the goat 4 units, in the mouse 13 units, in the rabbit 2000 units, in the fowl 200,000 units. This table applies, as already mentioned, only to subcutaneous and intravenous, but not to intra-cerebral injection. It has long been known that the hedgehog, the pig, the ichneumon, and many snakes are very resistant to snake venom, and that scorpions are resistant to their own poison. Rats are very resistant to diphtheria toxin, and mice are but little susceptible. Crocodiles are very resistant to many bacterial poisons, and especially to tetanus toxin, being susceptible, however, to diphtheria toxin. The supposition that resistance to poison is due in all these instances to the presence of antitoxins in the animal species in question is on the whole erroneous (Vaillard); such a condition occurs only in exceptional cases, for instance, in scorpions (Metschnikoff). After the injection of tetanus toxin into turtles their blood remains poisonous for several months, *i.e.* capable of inducing tetanus, although the animals themselves are absolutely resistant. This is accounted for by the absence in this particular species of animal of cells and groups of cells susceptible to poison.

The existence in the different animal species of these fundamental differences in regard to resistance to poison must be taken into consideration in attempting to explain general natural power of resistance. There are cases in which it is only because of the pre-existing resistance to poison that the phagocytes and alexins are enabled to exert their protective powers. Resistance to poison must therefore be regarded as an important factor in general natural power of resistance.

II. Specific Immunity

As a result of recovery from a specific disease or of protective inoculation.

A. Specific Immunity *as a result of recovery from specific disease.*—Specific protection, lasting for a comparatively long time, is conferred after recovery from an acute eruptive fever such as small-pox, scarlatina, or measles, and also after typhoid fever; and in animals after recovery from cattle plague, contagious pleuro-pneumonia of cattle, sheep-pox, contagious pleuro-pneumonia of the horse, and black-quarter (G. *Rauschbrand*; F. *charbon symptomatique*).

There are other infective processes, however, which confer no protection, for instance, diphtheria, relapsing fever, and gonorrhœa, and after certain diseases, such as pneumonia and erysipelas, the individual appears to be even more liable to a subsequent attack. Malaria, too,

[1] The alcoholic bandage consists of eight layers of gauze bandage which has been dipped and rung out in 95 per cent alcohol (60 per cent alcohol is rarely employed) and applied directly to the skin. Over this is placed a layer of cotton wool, and then gutta-percha tissue, which is often perforated. To ensure success it is important that the bandage should cover a large area, for example, in cellulitis of the hand the whole forearm should also be bandaged. The arterial dilatation is due to the cutaneous irritation associated with the dehydrating action of the alcohol, hence propyl-alcohol is more efficacious than ethyl-alcohol, but the use of the former is not advisable, because of its unfavourable by-effects. One result of the locally increased arterial pressure is the disappearance of œdema, and alcoholic bandages are therefore beneficial in non-inflammatory swellings. Lastly, alcohol is of great benefit in caries of the teeth. They should be cleaned twice daily with a tooth-brush which has been well moistened in water, and been then dipped in alcohol, 45 per cent or weaker. After three or four weeks' treatment the caries ceases because of the increased arterial blood-supply to the pulp.

confers no real immunity, yet the risk of acquiring pernicious rapidly fatal malaria is diminished after residence for a considerable time in malarious districts. Asiatic cholera is as yet believed to confer protection of only short duration. In the case of those specific infective processes which generally confer immunity, a slight attack affords as good a protection as would a severe one. This is probably the reason why, in the case of endemic infective diseases, such as typhoid fever or yellow fever, the inhabitants are usually less endangered than strangers, the former having probably previously suffered from the infection in a mild form.

In those instances where the blood serum of human patients who have recovered from cholera or typhoid fever has been examined, the same protective substances have been hitherto found as in animals which have been experimentally infected with cholera vibrios or typhoid bacilli. It is therefore advisable to deal under one heading with the conditions found in specific immunity in spite of their different modes of origin, for the specific immunity is essentially the same in both cases. It is always due to an *absolutely specific* protection against a certain infective agent or its poison, and is not due to a general increase of resistance. Not only so, but recovery from a specific disease may be followed by increased susceptibility to other infective diseases; thus a severe attack of measles is not infrequently the starting-point for tuberculosis.

B. Protective Inoculation (active or isopathic immunisation).—Since the discovery of antitoxins and antitoxic sera by Behring, two methods of artificial specific immunisation have been recognised: (1) *Protective inoculation*, whereby the specific protective substances have to be formed in the body itself; (2) Immunisation by transference of protective substances previously formed in *antitoxic serum*. Ehrlich designated the former method as *active*, the latter as *passive* immunisation. Behring has, however, of late preferred to term the former *isopathic* and the latter *antitoxic* immunisation. The chief difference between the two is as follows:—In isopathic immunisation, true protective inoculation, the individual must undergo a reaction, often a feverish one, after the introduction of the infective material. The antitoxin is meanwhile being formed, and protection is therefore obtained only after a period of time. The resulting specific immunity will, however, persist for a long time, usually for months, because the toxins which have been formed become bound to the tissue elements, and even after repeated severe venesections are not entirely got rid of in the withdrawn blood. In contradistinction hereto antitoxic immunisation by the introduction of serum induces specific protection at once without the production of any morbid symptoms; but the immunity conferred is of very brief duration (8 to 14 days), especially if blood serum of another species of animal be employed, for, being a foreign substance, it will be rapidly excreted. Such immunity, however, is of longer duration after the use of serum of the same species—in horses, for example, on using tetanus serum obtained from horses.

Just as in natural resistance we have to distinguish between resistance to bacteria and resistance to poison, must we do the same when dealing with specific immunity, and must therefore differentiate between "specific immunity to bacteria" and "specific immunity to toxin." Immunity to diphtheria is exclusively of the latter kind; diphtheria antitoxic serum has no action upon living diphtheria bacilli, it merely neutralises their poison. Diphtheria bacilli can even be cultivated in the specific curative serum. Diphtheria bacilli in a patient's body have to be overcome through the agency of the natural power of resistance, and this is easily effected if the toxins are rendered impotent by injection of antitoxin. The same holds good for tetanus. Behring, nevertheless, has recently advocated that there should also be local treatment of diphtheritic membranes, and especially those of the nose, with curative serum; and likewise in tetanus that the tetanus serum, or at least a portion thereof, should be injected as near as possible to the seat of infection if it be known.

If there be specific immunity to bacteria on the other hand, such as is induced by treating animals with cholera or typhoid bacteria, the serum of the immunised animal is merely bactericidal to the bacterium in question, and has no action on its integral cell poison. Hence if an animal previously treated with cholera vibrios be afterwards infected with a large number of the same, the latter will indeed perish, but a fatal intoxication may be induced by their cell poisons. This is one of the chief reasons of the failure to obtain an efficacious serum suitable for the treatment of cholera and typhoid in the human subject. Another point of importance is that in specific immunity to poison the condition is often a regional one, analogous to the condition dealt with when speaking of natural resistance to poison. Rabbits which, by means of long-continued treatment with bouillon containing the poison of tetanus, have become thoroughly immune to subcutaneous inoculation of tetanus toxin, are killed by intra-cerebral inoculation (Roux and Borrel). The conditions are similar in many other instances; for example, guinea-pigs in which a high degree of specific immunity has been induced by treatment with cholera vibrios are nevertheless not protected against intestinal infection with virulent cholera vibrios.

The following methods of active or isopathic inoculation may be differentiated:—

(1) Protective inoculation with living virulent virus.

(2) With living yet attenuated virus.
(3) With dead virus.
(4) With the bacterial cellular substances of the same.
(5) With dissolved toxic products of bacteria.

1. *Protective Inoculation with Living Virulent Virus.*—Inoculation for small-pox, which consists in the transference to healthy individuals of the contents of the variola pustule, was introduced into England in 1871 by Lady Mary Wortley Montagu, wife of the English ambassador at Constantinople, and was much used in England, Germany, and other countries in spite of its inseparable danger. Inoculation often caused severe or even fatal small-pox, and the disease was thus spread. The inoculated disease was, however, usually of a mild form, probably because inoculation into the skin affords conditions less favourable for the development of the small-pox virus than does natural contagion, in which instance the agent is probably carried from the mucous membranes into the blood and then multiplies there.

There has been much said of late regarding protective inoculation by means of subcutaneous injection of bouillon cultures of living virulent cholera vibrios, such as had been tried on a large scale in Spain by Ferran in the eighties. Cholera vibrios cannot multiply in the subcutaneous tissue; they die, are dissolved, and absorbed. In Ferran's inoculations the methods were imperfect and his statistics are quite unreliable. Haffkine, working much more accurately, has lately employed this method extensively in India, yet he generally uses dead cultures, and the subject is therefore discussed on p. 352.

In contagious pleuro-pneumonia of cattle, protective inoculation with the living virus (which has not yet been isolated) has been proved to confer effective protection, and is employed practically. Inoculation is performed at the end of the tail by subcutaneous injection either of lymph or fluid from the pulmonary tissue of a newly-killed animal suffering from the disease (" warm lymph "). The first result is swelling and inflammation resembling erysipelas around the seat of inoculation. The change often affects the whole tail, or extends even farther. There is in addition not infrequently necrosis and loss of the end of the tail, and also some feverish reaction. Animals thus treated prove immune to artificial and to natural infection of pleuro-pneumonia.

2. *Protective Inoculation with Living yet Attenuated Virus.*—In 1878 Buchner gave proof of the inconstancy of the anthrax bacillus when cultivated under definite conditions outside the body, but this inconstancy was not at that time made use of to confer protection. It is to the genius of Pasteur that we owe our knowledge of systematic protective inoculation by means of artificially induced attenuation. Pasteur's first success was gained in the disease termed fowl cholera (avian septicæmia); old cultures of fowl cholera bacilli, which have been exposed to the air and thus attenuated, were injected into the pectoral muscles of fowls, and induced a process of local inflammation and necrosis, which was followed by general specific immunisation of the birds.

The subsequent means of attenuation were the following:—

(*a*) *Attenuation by high temperature.*—Toussaint in 1880 demonstrated that anthrax blood loses its infective power by being heated to 55° C. for ten minutes; and in the following year Pasteur published his method of protective inoculation against anthrax, which is also founded on the power possessed by a high temperature (42°-43° C.) of attenuating anthrax bacilli. At this temperature anthrax bacilli can multiply, but cannot sporulate. Inoculation is commenced with the weaker cultures, which have been kept for twenty-four days at 42°-43° C. (first vaccine), and ten to fourteen days later a second and final injection is given, using the more powerful cultures which have been kept for twelve days at the above temperature (second vaccine). Animals thus treated are immunised against virulent anthrax. This method was stoutly opposed by R. Koch, yet was soon extensively employed in France and Russia, and afterwards in other countries, and favourable results were obtained in cattle and still more so in sheep. In Hungary, for example, according to Hutyra, 16,082 horses, 210,750 head of cattle, and 1,118,443 sheep were inoculated against anthrax from 1889 to 1893. The total loss in the first three years amounted to 2·18-2·44 per cent, and was only 0·61-0·55 per cent in the last two years. Opinions differ, however, as to the efficacy of this method, for the constancy of the vaccine is sometimes variable, and inoculation may therefore either confer no protection or may induce the disease and cause loss of stock. Again, the protection conferred even in the most favourable case is only of about one year's duration. Hence these protective inoculations should only be used in districts where anthrax is endemic and causes serious loss.

Arloing discovered a mode of protecting against *black-quarter* (G. *Rauschbrand*) which has proved of practical value. The dried and pulverised muscle-substance of affected animals is attenuated by heating to 100° C., is then mixed with sterile water, and injected into young cattle. It is usual to employ a weak infective material twice, and then a more powerful one. Favourable results were obtained in Germany (in Baden more particularly), Austria, and Switzerland. Kitt first modified this method by attenuating with steam under ordinary pressure, and afterwards by the use of pure cultures of Rauschbrand bacilli, which, on being subcutaneously inoculated into sheep and cattle, do not induce the disease, yet do confer immunity.

According to Kitt's statistics, the loss of stock from black-quarter was as follows :—

	Uninoculated.	Inoculated.
1886 . .	107·5 per 1000	5·6 per 1000
1887 . .	43·4 ,,	9·6 ,,
1888 . .	78·8 ,,	11·1 ,,
1889 . .	30·6 ,,	17·2 ,,
1890 . .	33·8 ,,	2·5 ,,

In the year 1890, out of 1167 cattle inoculated once with Kitt's vaccine there was only one death, and this was attributed to the inoculation, whilst of 2803 head of cattle which had not been inoculated 44 died.

(*b*) *Passage through less susceptible Animal Species.*—An important step was gained when Pasteur achieved attenuation by means of *passage* through less susceptible animal species, as was first done in swine erysipelas. The principle was essentially the same as that of Jenner's vaccination, but this fact was not so clearly understood then as it is now. In 1789 Edward Jenner published the fact, which had long been popularly known in Gloucestershire, but which he thoroughly investigated in his own native place, that recovery from cow-pox affords protection against small-pox, and he also showed that cow-pox may be artificially transferred from one individual to another without losing its protective power. But there is now abundant proof that cow-pox is nothing but the variola of man attenuated by transference to the ox or calf. Jenner's vaccination soon came into general use, but it was then found that the protection conferred only lasts about ten to twelve years. The protective power of vaccination was proved by Jenner himself and his contemporaries by thousands of experiments with subsequent inoculation of small-pox. Another most convincing proof in favour of vaccination is found in the trivial mortality from small-pox in those countries in which, as compared with others, vaccination has been enforced by law. The experience gained in the Franco-Prussian war of 1871 was almost equivalent to that of an experiment on a large scale, for the German, in contradistinction to the French troops, had been vaccinated and for the most part revaccinated. The latter, during the campaign, lost 23,000 men from small-pox, whilst the Germans only lost a few hundreds, although their whole army was in France, where an epidemic of small-pox was raging among the civil population.

Now Pasteur, in the case of swine erysipelas, showed that the bacilli taken from the pig gain in virulence on transference to pigeons, but that they become less virulent after repeated *passage* through rabbits, and that they are then suitable for protective inoculation of swine. The last *passages* yielded vaccine No. 1, then twelve days later followed inoculation with the bacilli of pigeon erysipelas (vaccine No. 2). The animals are then highly immune, and *Der land- u. forstwirtschaftliche Kongress* at Vienna in 1890 warmly advocated the use of protective inoculation. It has been tried, more especially in Hungary, and the disadvantages of the method have since then become evident. Deaths from the disease are common among the more susceptible breeds of swine, and young swine which have been inoculated appear unsuitable for fattening. There is also a danger of swine erysipelas spreading because of the inoculations. Preference has, therefore, since then been given to other methods.

(*c*) *Attenuation by drying the Vaccine.*—The virus of rabies is not yet known, yet Pasteur's experiments, brilliantly conceived and patiently carried through, furnished us with means of prophylaxis and cure, which have long been recognised as of the greatest benefit to mankind. Pasteur first found that the virulence of rabic virus is exalted by successive subdural *passages* through rabbits, whilst weakened by *passage* through apes. He strove to make use of this latter fact in his attempts to immunise dogs, but as the results obtained were so inconstant he forsook this apparently hopeful line of research for a new method of attenuation, that by drying.

By drying in an atmosphere freed from water and carbonic acid he succeeded in attenuating the highly virulent spinal cords of rabid rabbits, in which the incubation period has been shortened from about fourteen to seven days by numerous *passages* ("fixed virus"), and he was able to attenuate the cords to any degree proportionate to the duration of the desiccation, or even to render them completely inactive, and thus to transform them into a vaccine. Animals which were at first inoculated with very weak virus could withstand in succession the stronger, and lastly the most virulent spinal cords without becoming diseased. Pasteur then employed this method for the treatment of persons bitten by rabid dogs, wolves, etc., for he rightly supposed that a protective inoculation may be possible and successful even subsequent to infection, if the infective process be at first a localised one and pursue a chronic course. The Pasteur treatment of rabies is therefore not a true curative method, but essentially an active immunisation. To attenuate the spinal cord by desiccation it is kept at 22° C. in a vessel, the bottom of which is covered with fragments of caustic potash. A cord kept here for one to four days still causes rabies within seven days; by the use of a cord dried for more than five days the incubation period is prolonged, and a cord dried for twelve to fourteen days has become inactive. The treatment of the human subject commences with the injection of 1-3 cc. of an emulsion of completely attenuated cord, which is prepared by the trituration of 1 cm. of cord in 5 cc. of indifferent fluid; and within twenty days gradually increasing the strength of the dose till a cord dried for two days is used.

Complete immunity is only attained three to four weeks after commencement of the treatment, hence it is important to begin the latter as soon as possible. Deaths from rabies after the treatment has been carried out cannot consequently be included in any statistics unless the treatment has been completed at least fourteen days previously, for complete protection can only then be hoped for. The treatment has been gradually rendered more simple and efficacious, for instead of injections being often repeated and the degree of virulence slowly raised, fewer injections and a more rapid increase of virulence are now employed. The results in consequence are gradually becoming more favourable, and between 1886 and 1890 the returns from the Pasteur Institute in Paris are as follows:—

1886,	treated 2617;	died,	25 = 0·94	per cent.
1887	,, 1770;	,,	13 = 0·72	,,
1888	,, 1622;	,,	9 = 0·55	,,
1889	,, 1830;	,,	6 = 0·33	,,
1890	,, 1540;	,,	5 = 0·32	,,

As regards the locality of the bite, on which depends the danger of infection, the statistics of the same Institute for 1890 are as follows:—

Bite on head,	mortality,	0·85	per cent.
,, hand,	,,	0·45	,,
,, limbs and trunk,	,,	0·	,,

Institutes of preventive medicine formed after the model of the Pasteur Institute in Paris have been gradually founded in every country, and recently also in Germany, where in consequence of police regulations rabies is comparatively rare. The results are everywhere very favourable, and the average mortality is less than 0·2 per cent.

(*d*) *Attenuation by the Addition of Antiseptics to the Nutrient Medium.*—Virulence can be reduced by the cultivation of infective agents on media to which a weak antiseptic has been added. The sporulation of anthrax bacilli can be permanently inhibited by growth in carbolised bouillon, hence the bacilli can be attenuated to any extent. Such methods possess as yet no practical interest.

3. *Protective Inoculation with Killed Cultures.*—In many cases the same degree of immunity may be conferred by the use of dead cultures as by the use of living cultures of the virus, and the method is safer and simpler. Such is the case in cholera, typhoid fever, and plague.

(*a*) Cholera.—Protective inoculation against cholera originated in R. Pfeiffer's discoveries regarding the immunisation of guinea-pigs by treatment with cholera cultures. Living cholera vibrios are injected into the peritoneum of such an immunised animal, and drops of the exudation are from time to time withdrawn by glass capillary tubes and examined in either a fresh or stained condition. The vibrios are seen to lose their motility almost immediately, after ten minutes they swell up, and ten minutes later break up into little balls resembling micrococci, which after another twenty minutes can no longer be seen, and have apparently been completely dissolved in the peritoneal exudation. The same process is seen if the serum of the immunised animal or the blood serum of a patient convalescing from cholera be mixed with living cholera vibrios and injected into the peritoneum of a normal guinea-pig. The normal animal now acts like an immunised one, the cholera vibrios soon die, and the animal remains alive whilst control animals succumb (Pfeiffer's reaction). As the destruction of the vibrios is a rapid one only when the animal has been treated with *true* cholera vibrios, and not merely with those which are similar, yet specifically different, this reaction can *vice versa* be employed for the diagnosis of true cholera vibrios provided we have a supply of true cholera immune serum.

Guinea-pigs may be immunised not only by living cholera vibrios, but also by the use of cultures which have been killed either by heating to 56° C. for one hour, or by chloroform vapour. In the protective inoculation of man the employment of dead cultures, to which 0·5 per cent of carbolic acid may be added without impairing their activity, is obviously of great advantage.

The substances which are called into action in the serum of the immunised animal body were termed "*bacteriolytic*" or "*specific bactericidal*" substances by R. Pfeiffer, to distinguish them from the bactericidal alexins of normal serum, for the action of the former is strictly specific, *i.e.* directed only against cholera vibrios. This immune serum has no antitoxic action whatever on the poisons contained in the cholera vibrios. The action is merely an antibacterial one leading to destruction of living cholera vibrios, whilst it is always possible, by large quantities of a virulent killed cholera culture, to *poison* an animal immune to cholera, as already mentioned.

Cholera immune serum can act even in extreme dilution. The degree of potency is tested by mixing a given quantity of serum with five to ten times the minimum lethal dose of an agar culture of virulent cholera vibrios, and injecting the mixture into the peritoneum of guinea-pigs. Pfeiffer's standard is the smallest quantity of serum which in one hour and within the peritoneal cavity will dissolve that quantity of vibrios, the weight of the guinea-pig employed averaging 250 grammes. It is now definitely ascertained that as regards the bacteriolytic substances of the immune serum we have, as a matter of fact, to deal with two different substances, the joint action of which leads to the dissolution of cholera vibrios. Such a result may be seen not only in the peritoneum of guinea-pigs, but also outside the body in reagent glass or hanging drop, if we employ

absolutely fresh immune serum, or, to one no longer so, add a drop of fresh normal serum (Bordet). Pfeiffer showed that even on the introduction of old and hence apparently inactive serum, or of immune serum which had been heated to 60°-65° C., into the peritoneum of a normal animal, vibrios are dissolved there. This was formerly regarded as due to a sort of "regeneration" of the true specific anti-substance which is preserved in spite of heating to 65° C., but we now know that the phenomenon is due to the presence of the normal alexins and to a combined action. By the action of the specific anti-substances, retained after heating to 65° C., the cholera vibrios become predisposed to the solvent action of the alexins. The French, therefore, often designate this anti-substance as "substance sensibilitrice" (*v.* p. 370).

The formation of these specific anti-substances in the animal body has hitherto been shown to occur chiefly in the spleen, bone-marrow, and lymphatic glands (Pfeiffer, Marx, Wassermann).

After these discoveries had proved the possibility of conferring to men and animals definite protection against cholera, Haffkine in India carried out similar protective inoculations with cholera vibrios in more than 40,000 persons.

Emulsions of agar cultures of living cholera vibrios are injected subcutaneously. His first vaccine is $\frac{1}{12}$th of an agar culture of dead vibrios, or of vibrios which have been attenuated by cultivating a series of agar subcultures at 39° C. The second vaccine, $\frac{1}{12}$th of an agar culture of vibrios rendered virulent by *passage*, is afterwards injected. A few hours after the injections the temperature usually rises 1°-2° C., but returns to normal in twenty-four hours. Such a reaction with local pain and swelling, with rigor, depression, and anorexia, is, however, not an effect peculiar to cholera vibrios, but follows upon the subcutaneous injection of all bacterial emulsions, and even of innocuous and killed bacteria (Buchner, 1890). Kolle then demonstrated that one injection of a dead cholera culture—2 mg. fresh agar culture suspended in 1 cc. of bouillon—causes the same effect as repeated inoculations, and that the bacteriolytic power of the serum beginning to rise after the fifth day reaches its height on the twentieth. Protection due to preventive inoculation may therefore be looked for after the fifth day, an expectation which harmonises with actual results so far.

The results of preventive inoculations are on the whole favourable, even though, owing to the existing difficulties, exact statistical proof of the total morbidity and mortality of inoculated and non-inoculated be not forthcoming. Yet there are many observations indicating its value. In Calcutta in 1894, a record was made of every case of cholera occurring in thirty-six houses with a total of 521 inhabitants: 181 had been inoculated before the outbreak of the epidemic, some a considerable time previously, and of these 4 (2·2 per cent) were attacked and died of cholera; 340 had not been inoculated, and 39 of them (11·6 per cent) died. In one of these houses there were 18 inhabitants; of the 7 uninoculated persons 4 were attacked and 3 died, and not one of the 11 inoculated individuals was attacked. During the cholera epidemic of "The Gya Jail" inoculation was only commenced after the beginning of the outbreak. In 287 uninoculated persons the final mortality was 4·9 per cent, being 2·4 per cent among the inoculated. We must assume that these protective inoculations, to a certain if not a great extent, do not act by an accumulation of specific anti-cholera substances in the body, but by an increase of natural general resistance. That this assumption is correct is also indicated by Haffkine's observations, namely, that protection does not last long, and has at any rate completely vanished after fifteen months.

A very effective anti-cholera serum can be obtained from animals—guinea-pigs, rabbits, and goats—by treatment with dead cholera vibrios. It is indeed of no value in the treatment of cholera, but is so for the differential diagnosis of cholera vibrios from other closely related forms. Goats yield a particularly potent serum, but rabbits are most suitable, for even on the fifth day after a single injection of about three agar cultures, previously killed at 60° C., they yield a serum which even in solution of 1-200 is suitable for diagnostic purposes. The diagnosis of cholera vibrios by means of such a cholera immune serum is made either by observing Pfeiffer's phenomenon, *i.e.* the rapid dissolution of vibrios introduced with immune serum into the peritoneum of a healthy animal, or more simply by the specific agglutination discovered by Gruber and Durham.

The phenomenon of *agglutination* consists in the fact that cholera vibrios brought into contact with cholera immune serum in a test-tube lose their active motility, and, adhering to one another, collect into little clumps. The clumps soon become larger, and form first small and then larger flakes, which are deposited as a sort of sediment at the bottom of the fluid, which now becomes clear. Other observers had previously seen similar phenomena, but had not recognised their diagnostic significance. Gruber and Durham were the first to describe agglutination accurately, and to regard it as a fundamental action of specific bacteriolytic immune serum, and their statements were immediately afterwards confirmed and amplified by the writings of R. Pfeiffer and Colle, and Pfeiffer and Vagedes.

Agglutination is not only seen with cholera vibrios. If animals have been treated with certain forms of vibrios, typhoid bacilli, B. coli

or B. pyocyaneus, their blood-serum even in very small quantities will, according to Gruber and Durham, respectively agglutinate these different bacteria in suspension. The phenomenon can either be observed macroscopically in tubes or microscopically in hanging drops, the latter being the finer method of the two. In Gruber's reaction, in short, the different bacteria are thrown down by their respective specific immune serum. Yet, according to Gruber, the reaction is not an absolutely specific one, since powerful cholera immune serum, for example, can also agglutinate other allied forms of vibrios. Yet an immune serum always acts most effectively (*i.e.* is still potent when greatly diluted) on its respective specific bacterium. It is still doubtful whether the agglutinins—those substances of the immune serum which cause surface adhesion of bacteria—are identical with the true specific anti-substances of the immune serum, or whether both substances exist independently in the serum. The agglutinins, at any rate, as compared with the alexins, exhibit the same resistance to a temperature of 60° to 65° C., to putrefaction and to the influence of light as the true specific anti-substances, and therefore closely resemble the latter. Cases, however, have been observed where the serum possessed distinct power of agglutination, yet no specific protective power.

A point of importance is that the agglutinins disappear, or are used up during the reaction (Gruber), just as occurs if bacteria are cultivated in their specific agglutinating serum (Pfeiffer). In both cases the agglutinins are directly absorbed by the specific bacteria, as has been lately proved by the fact that the absorbed agglutinins can be redissolved from the bacteria by dilute alkalies, and separated by centrifuging (M. Hahn and Trommsdorff). The agglutinin solution thus obtained can again agglutinate specific bacteria added to it.

(*b*) Typhoid.—The immunisation of animals against typhoid bacilli is performed in the same way as against cholera vibrios (R. Pfeiffer). Agar cultures of typhoid bacilli, one day old, are suspended in bouillon, are killed by heating at 65° for one hour, and are once or repeatedly injected. The serum of the animal thus treated then exhibits a specific bactericidal action on typhoid bacilli, *i.e.* if living typhoid bacilli are injected together with this immune serum into the peritoneum of a normal guinea-pig they are rapidly destroyed, whilst without the immune serum the same bacteria would multiply and cause the death of the animal. Such an experiment is still successful if the specific immune serum has been heated to 60°-65°. The true active anti-substance is therefore preserved in the typhoid immune serum, even at the above-mentioned temperature, and the normal animal body has only to supply the requisite alexin, whilst the specific anti-substance predisposes the bacteria to the influence of the latter. The conditions are therefore analogous to those of cholera.

In the human subject a certain degree of protection can be conferred against typhoid as against cholera, and the serum then gains distinct specifically bactericidal power. A single injection of 2 mg. of a dead agar culture ($\frac{1}{10}$th of a culture), to which 0·5 per cent of carbolic acid has been added, is sufficient for an adult. Injection under the skin of the back is followed by transient elevation of temperature to 38·5° C., headache, and depression; the seat of injection and the neighbouring lymphatic glands are for a few days very tender to pressure. The blood, even a week afterwards, may act specifically on typhoid bacilli, for when such serum even in a dilution of 1-200 is mixed with typhoid bacilli and injected into the peritoneum of guinea-pigs the bacilli are quickly destroyed. Such serum, however, does not always agglutinate *in vitro.* It is not yet known whether such inoculation affords the human subject protection from typhoid when infection has occurred in the usual manner *per os.* Soldiers on their arrival in India have of late been inoculated for typhoid by Haffkine, but the results are not as yet decisive.

Treatment of animals, guinea-pigs, rabbits, and goats does not furnish a serum as active against typhoid bacilli as was the case in cholera. Yet rabbits after a single injection of three killed agar cultures usually furnish a serum which agglutinates in a dilution of 1-150. The power of agglutinating typhoid bacilli possessed by the serum of animals treated with typhoid bacilli, or of persons who have recovered from typhoid, is of great importance for the differential diagnosis of these bacilli. The phenomenon is the same as in the case of cholera (Gruber and Durham). The reaction is, however, not *absolutely* specific, for typhoid immune serum agglutinates not only true typhoid bacilli, but also B. coli and B. enteritidis Gaertneri. But whilst true typhoid bacilli are agglutinated by a powerful typhoid immune serum, even in a dilution of 1-100 or 1-500, a more concentrated serum—a dilution not greater than 1-30 or 1-50—is always necessary to agglutinate these other bacteria. The phenomenon of agglutination acquired great practical importance from the clinical standpoint by Widal's discovery that typhoid bacilli are agglutinated not only by the serum of persons who have recovered from typhoid, but also as a rule by that of typhoid patients in the first few weeks or days of their illness. This method of diagnosis ought to be termed the Gruber-Widal reaction, for it is merely the clinical application of the phenomenon of agglutination first discovered by Gruber.

The Technique of the Gruber-Widal Reaction.—The serum of the suspected typhoid patient

is mixed with typhoid bacilli, which have been grown on agar and are suspended in bouillon. The result is watched at 37° C. either in a tube or hanging drop. The blood used is obtained by puncturing the tip of the finger or lobe of the ear, is drawn into a sterile pipette, and then blown into a small narrow sterilised tube, which is laid horizontally till coagulation begins and then placed upright. In a few hours sufficient serum will have collected at the bottom of the tube, or the separation may be hastened by centrifuging. The serum should then be at once diluted with 50 parts of bouillon, for normal serum in a concentration greater than this will sometimes agglutinate. The serum of typhoid patients will usually agglutinate when greatly diluted (up to 1-5000), but a marked agglutination with 1-50 is sufficient for diagnostic proof, provided that it occurs at about 20° C., within one or at most two hours. The typhoid culture employed must not be more than twenty-four hours old and of moderate virulence. The reaction is characterised by the formation of first smaller and then larger clumps, whilst a control test with the same typhoid emulsion does not show this phenomenon in the same period of time.

The serum of typhoid patients sometimes gives this reaction on the second or third day, but usually only later and sometimes only during convalescence. The agglutinative power may, however, persist for a long time after recovery from typhoid, even though the attack be mild, and this may give rise to errors in diagnosis. That there is really a fresh attack of typhoid may be proved by observing on repeated quantitative determination that the agglutinative power is increasing. The severity of an attack cannot be deduced from the intensity of the agglutination. At the height of the disease the agglutinative power may sometimes be reduced; and in a mild attack the serum may sometimes agglutinate very markedly. The negative result of one examination does not exclude the possibility of typhoid; examination should be again undertaken, as it is often only in the later stages that the serum has the power of agglutinating.

The agglutinative power of a typhoid serum is usually but not always proportionate to its specific bactericidal action on typhoid bacilli. Thus the serum of dogs after the introduction of typhoid bacilli by the mouth may agglutinate and yet have no specific bactericidal power, which it does, however, possess if the typhoid bacilli have been inoculated into the peritoneum.

(c) Plague.—By the repeated intra-venous or intra-peritoneal injection of plague cultures killed at 58° a serum can be obtained from rabbits. Small doses of this serum protect other rabbits against virulent plague bacilli, even though used twelve hours subsequent to infection (Yersin, Calmette, and Borrel). Yersin based his method for the production of plague serum on this fact.

In view of the fact that natural recovery from plague confers a definite, though only a relative, immunity to plague, Haffkine in 1897 tried direct active immunisation of man by means of killed plague cultures. The method consists in the subcutaneous injection of bouillon cultures of plague bacilli, one month old, killed at 70°. For an adult an injection of 2-3 cc. is made into the upper arm or abdomen, and is followed by swelling and tenderness at the seat of injection and by slight fever. This injection ought to be followed by a stronger dose after eight or ten days, but few people can be induced to submit to a second inoculation. During the plague epidemic in India Haffkine's inoculations have been performed on a large scale and with favourable results. When plague broke out in the prison of Byculla in Bombay, and after fifteen cases, eight of them fatal, had occurred between 23rd and 30th of January, Haffkine on the evening of the 30th of January inoculated 154 prisoners, who subjected themselves voluntarily, giving each one 3 cc. Up till the 6th of February there were only two cases of plague, neither being fatal, among these 154 persons situated in the same circumstances and living together with 177 non-inoculated. Of the latter, 14 were attacked and 6 died. During the plague epidemic of 1897 in the Portuguese town of Damaon, it is recorded that among 62 families the mortality from plague was 8 per cent in 250 inoculated individuals, and 29·8 per cent in 124 non-inoculated. In inoculated persons, moreover, the course of the disease is said to be milder and the buboes suppurate more rapidly. Further statistics have also indicated the remarkable reduction of mortality among the inoculated, and active immunisation with killed plague bacilli must therefore be strongly recommended, more especially for the protection of physicians, nurses, persons who have to carry out disinfection, and so on.

Immunity by this method is only obtained after about a week, as has been definitely proved by animal experiment, but it then lasts for months. According to the researches of the German Plague Commission, the immunising substances are contained in the cell substances of the plague bacilli, and not merely in the filtrates. The use of bouillon cultures is therefore of no special value, and it is better to use agar cultures which have been suspended in physiological saline solution, and then killed by being heated for one hour at 65° C. Heating to the boiling-point would destroy the immunising substances of the plague bacilli in contradistinction to those of typhoid and cholera bacteria. Before using this emulsion as a vaccine it is advisable to add 0·5 per cent of carbolic acid, which does not impair the power of the immunising substances. Larger quantities of plague bacilli can be injected into man than typhoid bacilli, for the former are less poisonous.

Plague immune serum obtained by the treat-

ment of animals with killed cultures has the power of agglutinating, and as a rule, but not invariably, that of immunising.

4. *Protective Inoculation with Bacterial Cell Substances.*—In different instances, for example, cholera, typhoid, and plague, we have seen that active immunisation may be effected by the killed bacterial cells, whilst the associated filtrates are inactive. In these instances, therefore, the substances conferring immunity must be contained in the bacterial cells themselves. The bacterial cell substances, which are mainly of a proteid nature, are obtained—(*a*) By extraction with boiling water, whereby albumoses—so-called bacterial proteins—are formed (the older tuberculin, mallein); (*b*) or without induction of chemical change by purely mechanical pulverisation of the bacterial cells (tuberculin TR, bacterial plasmin).

(*a*) *Immunisation with Bacterial Proteins—Tuberculin.*—Koch's tuberculin is obtained by concentrating at the boiling-point cultures of tubercle bacilli in glycerine bouillon. The proteids of the tubercle bacillus are in consequence partly dissolved, and they are purified by repeated precipitation with 60 per cent alcohol. The active principle, therefore, consists in the dissolved proteins of the tubercle bacillus which resemble albumose. Subcutaneously their action is similar to that of other bacterial proteins, causing hyperleucocytosis which in man is merely local, swelling, redness, tenderness, and with stronger doses fever, as previously mentioned. But as even a very minute quantity of tuberculin is sufficient to cause a reaction in the body, we assume that it has a specific action, as well as the general action of a bacterial protein manifested by increase of natural resistance to bacteria. Tuberculin has much less effect on healthy than on tuberculous guinea-pigs. The former can stand a subcutaneous dose of 1-2 cc., whilst the latter invariably succumb to 0·5 cc. Man is much more susceptible; in a healthy adult even 0·25 cc. causes great rise of temperature, severe local pain, general depression, and so on, whilst after 0·01 cc. the symptoms are as a rule only trivial. This dose given to a tubercular patient, however, usually causes severe fever, lasting twelve to eighteen hours, and at the same time a well-marked local reaction in tubercular areas, which is specially evident in tuberculosis of the skin, lupus, but which occurs in tuberculosis of the internal organs too. A few hours after the administration of tuberculin the tubercular areas commence to swell, and there is marked increase of blood-supply within them and in their immediate vicinity.

In consequence of the great difference in the susceptibility of tubercular and of healthy people to tuberculin, its use is of considerable importance for the diagnosis of latent tuberculosis. The normal temperature having previously been accurately ascertained, an adult receives a first injection of 1 mg. tuberculin, a few days afterwards 5 mg., and still later 10 mg. The corresponding doses for children under 10 years of age are 0·5, 1·0, and 5·0 mg. The temperature must be taken every three hours, and an elevation of 0·5° C. above the normal temperature of the individual indicates a positive reaction. This diagnostic procedure ought to be employed more than it has hitherto been. If the reaction be doubtful the same dose is to be repeated, or a larger one may be given. No injection should ever be given to a feverish patient. Tuberculin remains unchanged for a long time because it contains a large amount of glycerine.

In veterinary medicine tuberculin has for years been in general use as a diagnostic agent, and especially for the diagnosis of tuberculosis in cattle. The dose for a cow is 0·5 cc., and it is only a greater rise of temperature than 1° C. which can be regarded as positive proof of tuberculosis. There will be few errors in diagnosis if the diagnostic inoculations and the post-mortem examinations of the animals are accurately and carefully made.

Out of 124 animals which had previously reacted to tuberculin Nocard found 123 to be tuberculous, although the disease was often localised and obscure. These diagnostic inoculations have been much used on a large scale in Denmark and France, and have been an excellent means of weeding out tuberculous animals.

R. Koch recommended tuberculin for therapeutic as well as diagnostic purposes; at first it was taken up enthusiastically, but it was afterwards as severely discarded, yet the condemnation of tuberculin has probably been too pronounced. If guinea-pigs infected with tuberculosis are given an injection of dilute tuberculin every day, or every second day, their condition is improved, and death is deferred from the ninth to the nineteenth week. After death animals thus treated show marked pulmonary tuberculosis, whilst the tuberculosis of the abdominal organs is undergoing cure. Control animals not treated with tuberculin soon die from tuberculosis of the liver and spleen; pulmonary tuberculosis, which is a late manifestation after inoculation with tubercular bacilli, has not had time to develop in them. This favourable effect of tuberculin is not due to a direct action on the tubercle bacillus, but to the reaction—combined with hyperæmia—of the tissues which are the seat of tuberculosis. We have already indicated that tubercular lesions in an organ are cured by a permanent increase of blood in the latter. Hence tuberculin is merely a means of inducing local hyperæmia of tubercular foci.

In spite of these experimental results most physicians have had an unfavourable experience of tuberculin as a curative agent. This is due

to the fact that pulmonary tuberculosis in man, whenever it has been detected in consequence of fever, almost always represents a mixed infection with streptococci, and tuberculin treatment is unfavourable in the case of a streptococcal infection. But pure tuberculosis (simple tuberculosis) is similar to the artificially induced tuberculosis of guinea-pigs, and can be decidedly improved and not infrequently cured by tuberculin. Petruschky has lately reported many favourable cures. He recommends an intermittent method of treatment, *i.e.* regular alternation between two to three months' use of tuberculin and three to four months' pause. Alternation is necessary because tolerance to tuberculin is induced, and it therefore ceases to act. To effect a cure, it is said that treatment must on an average be continued for two years.

Mallein.—According to Nocard mallein is obtained by heating, by concentration to $\frac{1}{10}$th of its previous bulk, and by filtration of glycerine bouillon cultures of B. mallei one month old. Mallein is thus prepared in the same way as the older tuberculin, and it too belongs to the bacterial proteins. Opinions differ as to its value in diagnosis, for non-glandered animals sometimes react to mallein. Nocard considers that only those animals which yield a distinct reaction—a greater rise of temperature than 2°—in combination with definite clinical symptoms are to be regarded as affected with glanders, and therefore destroyed. The action of mallein, too, is not an absolutely specific one, for other bacterial proteins can cause similar although weaker effects.

(*b*) *Immunisation with Bacterial Cell Substances obtained mechanically.—Tuberculin TR.*—R. Koch's more recent tuberculin TR (1897) is obtained from virulent cultures of B. tuberculosis by drying in an exsiccator, by fine mechanical pulverisation and extraction with distilled water. The extract is divided, by centrifuging, into an upper fluid portion and a solid sediment. The latter, after being again dried, pulverised, and extracted with water, constitutes tuberculin TR in which glycerine induces precipitation, whereas the older tuberculin contains much glycerine.

According to Koch, tuberculin TR has the definite power of inducing specific immunity, which is said to be developed without any reaction. It is said that as soon as insusceptibility to tuberculin TR is gained there is also immunity to the older tuberculin and to the tubercle bacillus. Tuberculin TR, which, as well as the older tuberculin, is prepared at the manufactories at Höchst-a.-M., contains 10 mg. of solid substance in 1 cc., and for use is diluted with physiological saline solution. The first dose is usually 0·002 tuberculin, being equivalent to $\frac{1}{500}$th mg. solid substance. Injections are then given every second day in gradually increasing doses, care being taken to avoid any rise of temperature greater than 0·5° C. The dose is gradually increased till 20 mg. is given. With tuberculin TR, Koch was able to immunise animals, or to cure those previously infected. But tuberculin TR, as used in medical practice, has yielded results almost as unsatisfactory as those of the old tuberculin, and another disadvantage of the new preparation is that it can only be kept for a short time.

Bacterial Plasmin.—Mechanical pulverisation of the cells of the lower fungi, as a mode of obtaining their intra-cellular substances, had been employed by E. Buchner in the Hygienic Institute in Munich before Koch's publication on the tuberculin TR. It is this method which furnishes the main proof that the true fermentative substance, the zymase, is contained in a soluble form in the cell juice—plasmin—of beer-yeast cells. Pure yeast is mixed with infusorial earth and quartz-sand, thoroughly pulverised, and then exposed to hydraulic pressure of 300-500 atmospheres. The juice, which contains much albumin, is filtered and freed of all living cells, and it undergoes true alcoholic fermentation on sugar being added. We must assume that the zymase is a product of and is contained in the yeast cells, and that the fermentative energy of the yeast depends upon the production of zymase.

As the cell-juices or plasmins of the lower fungi can be obtained by this method in an unchanged and active form, M. Hahn, at the Hygienic Institute, Munich, therefore prepared the plasmins of cholera, typhoid, tubercle, and other bacteria, and employed them for immunisation. A single injection of 0·5 cc. of "cholera plasmin" appeared well adapted for immunising guinea-pigs against intra-peritoneal infection with ten times the fatal dose of living virulent cholera vibrios, and the immunity lasts at least three to four months and is specific. The action of "typhoid plasmin" is analogous. The sera of animals treated with either of these plasmins have a powerful specific agglutinative action. "Tuberculo-plasmin" after filtration is a clear pale yellow fluid containing nucleo-albumin, and it will keep for a considerable time after the addition of 20 per cent of glycerine and 5 per cent of common salt. This preparation has been used with favourable results in the treatment of guinea-pigs infected with tuberculosis, but in only a few cases has it been hitherto employed in the human subject.

5. *Immunisation with Toxic Filtrates of Bacterial Cultures.*—In 1886 Salmon and Smith showed that pigeons can be rendered immune to hog-cholera (hæmorrhagic septicæmia) by treatment with filtrates of hog-cholera microbes. Foà and Bonome, Beumer and Peiper, and lastly Charrin in 1887, performed similar experiments in the diseases of rabbits

artificially induced by proteus and B. pyocyaneus, Roux and Chamberland in malignant œdema, Roux (1888) in black-quarter (*Charbon symptomatique*), C. Fränkel in diphtheria, and so on.

It is especially in the latter cases that we are concerned with the production of a well-marked specific tolerance of poison. The nature of this tolerance was only made evident after the fundamental facts regarding bacterial toxins had been made known by Roux and Yersin. They showed in 1886, as a result of splendid research, that the poison of the diphtheria bacillus possesses special properties, and more especially extreme susceptibility to heat—being destroyed at 65° C.—and again the capability of being carried down mechanically by chemical precipitates (calcium phosphate) in the filtered cultured fluid, properties which had until then been recognised mainly in the digestive enzymes. Brieger and C. Fränkel confirmed and amplified these facts, and showed that the poisons of diphtheria and tetanus can be obtained in a moderately pure form by means of a method as simple as possible, *i.e.* from filtered cell-free cultures by simple precipitation with absolute alcohol. These poisons gave the reactions of albuminous substances, and were therefore termed "toxalbumins." But as Brieger has since said that they are not albuminous, it will probably be best to term them *specific toxins*, in contradistinction to the non-specific toxins resembling ptomaines, *e.g.* cadaverin, putrescin, etc. Specific toxins are characterised by the fact that they can cause in an animal all the symptoms of the infection in question, *e.g.* tetanus.

In regard to the origin of toxalbumins, Brieger and C. Fränkel believed that they were split off from the proteids of the nutrient medium by the action of the bacteria. This is not so, for if diphtheria and tetanus bacilli be successfully cultivated in nutrient fluids containing no proteid, toxins are still formed (Guinochet, Buchner). Hence the specific toxins must originate directly from the bacterial cells; they are products of the specific plasma of the bacteria, and are excreted during the life of the latter.

Now the specific toxins are to be regarded as the really active principle in the production of specific immunity to poison, although we have to note that solutions containing the poison alone are not suitable when starting immunisation (Roux and Yersin), and again that fluid cultures of diphtheria and tetanus which have lost their poisonous character will confer immunity. These phenomena are accounted for by the presence of modified toxins—which Ehrlich calls "toxones"—which are not themselves poisonous, yet bring about the formation of antitoxins. When an animal is to be immunised, it is advisable, the true poison being too dangerous, to make a start with its non-poisonous modifications which are contained along with modified poisons in the cultures.

After the discovery of diphtheria and tetanus poisons the first attempts at immunisation were unsuccessful as the animals died of chronic poisoning. Tolerance to poison was only gained after the filtrates had been weakened by heating to 60° C. (C. Fränkel) or after the addition of certain chemicals (*e.g.* iodine terchloride, Behring), and the toxins thus destroyed whilst the toxones remained. When once the animals had gained a certain degree of tolerance of poison, poisonous filtrates could then be employed in order to render the tolerance greater and more powerful.

Apart from diphtheria and tetanus attempts were afterwards made to isolate a soluble specific cholera toxin from cultures of cholera vibrios, and thereby to immunise animals to this poison (Behring and Ransom). Specific toxins were similarly isolated from typhoid bacilli, pneumococcus, plague bacillus, etc., but they have hitherto been of little practical value.

Direct active immunisation to poison is not generally applicable for practical purposes. This is partly because of its associated difficulty and danger, and still more so because in the case of diphtheria and tetanus we must confer rapid protection, and immunisation by injection of filtrates is but a very gradual process.

The great significance of active immunisation to poison by the use of toxic filtrates of bacterial cultures depends entirely on the discovery, made by Behring and Kitasato in 1890, of the antitoxic value of the serum of an animal immunised to poison (*i.e.* to diphtheria and tetanus toxin). It appeared that the protection from poison obtained by specific treatment can be transferred by the serum to other animals, and that they too are then immune to this specific poison (*passive* immunisation).

Just as immunity to toxic bacterial products can be induced, so can immunity to vegetable toxalbumoses be obtained by the use of these latter substances. By feeding with ricin, abrin, and robin, Ehrlich induced, even in the most susceptible animals, a high degree of immunity to these poisons, and similarly marked immunity to snake venom may be induced in various animals (Calmette, Physalix, and Bertrand).

C. Specific Immunity by the Transference of Blood-serum of actively Immunised Animals—Passive (Antitoxic) Immunisation—Serum Therapeutics.—The most rapid means of conferring specific immunity, and especially specific immunity to poison, is based on Behring's discovery, in 1890, of the transference of blood-serum of specifically immunised animals to other animals or man. It was in diphtheria that Behring first proved that the blood and serum of animals immunised to diphtheria is capable of at once rendering other animals insusceptible to what would otherwise

be a fatal dose of diphtheria poison, or, as he expressed it, "of destroying diphtheria poison."

In conjunction with Kitasato he afterwards proved the same for tetanus. The transference of immune serum in both cases first confers protection against the specific poison, and thus indirectly against infection with the living virus, for if their poisons are inactive both diphtheria and tetanus bacilli are overcome and destroyed by the natural power of resistance of the body. As the immunising anti-substances are contained in the immune serum they do not require to be formed in the new animal body; in passive immunisation, therefore, the new animal body does not require to undergo any reaction, for the effect is immediately induced. But another difference from active immunisation is the very short duration of the immunity conferred by transference of serum, for foreign serum does not remain long in the body, but is destroyed or excreted within eight to fourteen days. As compared with active immunity, therefore, passive immunity is only a transient one.

Passive immunisation has been performed not only in diphtheria and tetanus, but in a series of other infective processes, and has to some extent yielded results of practical value. Again, the active immunity to poison conferred by treatment with ricin, abrin, and robin can be transferred by the serum to other animals (Ehrlich). Ehrlich has also shown in these and other instances that it is not the serum, but the milk, which may be the vehicle for the transference of antitoxin, and that immunity may thus be transmitted from the mother to the offspring by suckling (*vide infra*). The transference of blood-serum does not only confer protection from subsequent infection, but, provided the conditions are otherwise favourable, it also induces a cure after precedent infection. It is experimentally proved that the resulting effect is essentially the same whether we inject a mixture of antitoxic serum and toxin, or inject each of them into different parts of the body, or lastly, whether we inject the serum twenty-four hours before the toxin. It is a very different matter, however, if the antitoxic serum be injected subsequent to infection. The greater the delay in the introduction of the serum the more unfavourable is the prognosis, the greater is the quantity of serum required, and the more uncertain is the result (*vide infra*).

1. *Mode of obtaining Antitoxic Serum.*—According to Behring the animals which are to furnish the serum (horses) are rapidly and safely immunised (*a*) by using unaltered bouillon cultures of tetanus bacilli—their filtrates are preferable—injecting $\frac{1}{20}$th of the minimum fatal dose at first and increasing to double this dose within four weeks; (*b*) or, as is better, by starting the treatment with larger quantities of cultures rendered less poisonous by the addition of suitable chemicals, such as iodine terchloride.

Behring gives the following special directions for the immunisation of horses: 200 cc. of filtered bouillon culture of tetanus, to which 0·5 per cent carbolic acid is added as a preservative, form the stock material. The toxic value of this culture must be such that 0·75 cc. suffices to kill with certainty an adult rabbit in three or four days. This carbolised culture is divided into four portions: (1) 20 cc. to which nothing further is added; (2) 40 cc. to which 0·125 per cent iodine terchloride is added; (3) 60 cc. to which 0·175 per cent iodine terchloride is added; (4) 80 cc. to which 0·25 per cent iodine terchloride is added. At the start 10 cc. of mixture No. 4 is subcutaneously injected into the horse, after eight days 20 cc., after another eight days 20 cc. again, and the remainder three days later. Mixtures Nos. 3, 2, and 1 are then consecutively employed, being administered gradually and at suitable intervals.

What Behring regards as essential in this method is not the mere use of cultures whose poison has been weakened by adding iodine terchloride (Vaillard uses solid iodine for the same purpose), but the subsequent employment of highly virulent cultures, or filtrates the virulence of which is unimpaired, for only thus can a degree of immunity sufficiently high to be of practical value be conferred. Cultures with unimpaired virulence had previously been employed by Emmerich and Vaillard for the production of a high degree of immunity. Behring's improved method is founded on the knowledge that the conferring of immunity is "not a sudden event, but a very gradual process." We may therefore infer "that the degree of immunity is capable of unlimited increase."

In the immunisation of the horse, even though the animal has been long under treatment, and has already been highly immunised, each fresh injection of tetanus bouillon gives rise to a reaction with fever, during which the immunising substances hitherto detectable in the urine disappear, and the urine may even contain tetanus poison (Behring and Casper, Brieger and Ehrlich). During this period no blood is to be taken from the animal for curative purposes. The former immunising value is not always regained until the eighth or tenth day, but it then undergoes gradual increase. Behring regards the appearance of the feverish rise of temperature during this period of reaction as a very favourable symptom, indicating that the poisons are being rendered innocuous in the body, and unless there be fever a further increase of immunity cannot be hoped for. The use of antipyretics in such a case is always to be avoided.

Immunisation against *diphtheria* is also best performed by the use of bouillon cultures of diphtheria attenuated by the addition of iodine

terchloride. Diphtheria cultures several months old are filtered through paper, and after the addition of 5 per cent of carbolic acid they remain in contact for two to three weeks with varying quantities of iodine terchloride (0·05-0·4 per cent). Guinea-pigs, sheep, and horses are immunised by the subcutaneous administration of such cultures in gradually increasing doses (Behring and Wernicke). Each dose of diphtheria poison—treated with iodine terchloride—must induce a local and general reaction. If the reaction be insufficient the immunising effect is very slight; if the reaction be too marked, leading to emaciation, immunisation is usually frustrated. Hence, in the process of immunisation there is much care and experience required, and it is important to weigh the animal from time to time, as a permanent loss of weight must be expressly avoided. Horses are the only animals employed for obtaining diphtheria antitoxic serum, both because they stand the treatment with toxins well, and because they are well suited for the removal of blood. Blood is very easily obtained from the jugular vein, and can always be obtained absolutely pure. The venesection wound usually heals so well that blood can be withdrawn fifty to a hundred times from the jugular vein of one horse. From the blood a clear serum is easily obtained, which when injected under the skin is quickly absorbed, and even in large doses is innocuous to man and animals. The diphtheria antitoxin is often separated from the serum by precipitation and employed as a dry preparation, which requires to be dissolved before being used. If the serum is to be maintained in a fluid form, however, 0·5 per cent of carbolic acid or a small piece of camphor (Roux) is usually added.

2. *Immunisation Value.*—For the correct employment of immune sera for prophylactic and therapeutic purposes it is, firstly, important to know that there is a definite quantitative relationship between the amount of antitoxin contained in serum and the amount of poison which the former is able to neutralise. The serum, therefore, can only bring about immunisation or cure according to the amount of antitoxic substance it contains. But in dealing with a pre-existing disease the amount of poison present in the body can never be accurately ascertained. A supply of antitoxic substance sufficient for the most unfavourable case must therefore be sought for. This is rendered possible by employing a serum of high immunising value, which contains the antitoxin in high concentration. To obtain such a serum the animal furnishing it must have been as highly immunised as possible. Behring has proved that those species of animals which are originally very susceptible to a given poison furnish a much more potent serum than, with the same degree of immunity, do those animals which have always possessed a considerable innate resistance to the poison. The reason for this is very obvious. It is not the natural innate resistance to poison, but only the resistance which is artificially induced by immunisation, which can be transferred by means of the serum. In order, therefore, to obtain a serum with a high immunising value those animal species must be employed which are naturally very susceptible. Such is the case in the horse as regards diphtheria and tetanus.

The activity of an immune serum, therefore, does not altogether depend on the degree of immunity of the animal from which it was derived, but on its immunising value. For the estimation of the latter (*i.e.* for the standardising of serum), the method commonly used in Germany till quite recently was the one elaborated by Ehrlich, Kossel, and Wassermann. Ten times the minimum lethal dose of a toxin is mixed in varying proportions in a reagent glass with the serum which is to be examined, the mixture is made up to 4 cc. with physiological saline solution, and then injected into guinea-pigs of 250, or at most 300 grammes weight. This method yields more accurate results than the former method of injecting toxin and serum into different parts of the body. It is important to take guinea-pigs of the same weight, as otherwise the results differ. The mixture in which, under such circumstances, the toxin is exactly neutralised—there is no change whatever induced in the animal—is used in estimating the immunising value. Behring and Ehrlich first prepared a *normal toxin.* This is the term applied to a toxin solution 0·01 cc. of which suffices to kill a guinea-pig of 250 grammes weight within five days. Hence normal diphtheria toxin (DTN^1) is a solution 1 cc. of which contains the minimum lethal dose for 100 guinea-pigs, each of 250 grammes weight, *i.e.* for 25,000 grammes of living guinea-pig. *A normal serum*, or normal diphtheria antitoxin (DAN^1), is a serum 0·1 cc. of which completely neutralises the lethal action of 1 cc. of normal diphtheria toxin; 1 cc. of this normal serum contains one *immunisation unit.* A serum 0·01 cc. of which suffices to neutralise the same amount of toxin is ten times normal (DAN^{10}), and if 0·001 is sufficient the serum is one hundred times normal (DAN^{100}); 1 cc. of this latter serum, therefore, contains one hundred units. Living cultures of diphtheria bacilli used sometimes to be employed instead of toxin solution for the estimation of the immunising value of a serum, but such a method is inadvisable as the results obtained are much less accurate.

In France the ordinary mode of estimation is as follows (Roux and Martin):—The amount of serum which just suffices to protect a guinea-pig from ten times the lethal dose of toxin is ascertained. The proportion between this amount of serum and the body-weight furnishes the value of the serum. For example, "a

serum whose immunising value is 10,000" signifies that an amount of serum equal in weight to $\frac{1}{10000}$th of the weight of a guinea-pig suffices to protect the animal completely against ten times the lethal dose of toxin. In the French method the body-weight of the animal is accordingly taken into account, and it is therefore not essential to use guinea-pigs of 250 to 300 grammes weight. Madsen, however, comparing the two methods, found that the German was the simpler and cheaper one.

Large numbers of immunising units (600-1500) are necessary when employing the curative serum in man. After diphtheria curative serum had become an article of commerce, it was found necessary in Germany to insist on a minimum number of immunisation units in order to protect the public from inferior preparations. The minimum first demanded was that 1 cc. of diphtheria curative serum should contain at least 100 units. Since 20th February 1895 the preparation of serum has been under State control, under which the serum is standardised, is proved to be free of deleterious substances, and the correct amount of antiseptic—carbolic acid—necessary for the proper preservation of the serum is added.

Ehrlich, the head of this serum-testing establishment, which is in connection with the *K. preussischen Institut für experimentelle Therapie* at Frankfurt-a.-M., has lately introduced a new and still more exact method of standardising serum. Diphtheria toxin and antitoxin in soluble form being somewhat variable, Ehrlich proceeds from an antitoxin dried by evaporation of the serum. This antitoxin is kept at the Frankfurt Institute in specially-constructed vacuum tubes, which each contain 2 grammes of dry antitoxin of 1700 units. Every two or three months one of these tubes is cautiously opened and its contents dissolved in 200 cc. of a fluid consisting of 10 per cent saline solution and glycerine. A serum seventeen times normal is thus obtained, and when diluted seventeen times 1 cc. contains exactly one unit. By the aid of this normal antitoxin a normal solution of toxin is then prepared by means of which again a given serum is standardised. It is by this method that diphtheria serum is now officially tested. The durability of the serum is also to some extent guaranteed by the fact that as each serum is tested a number of bottles are withheld, and their activity tested from time to time, and if it be found to diminish, all the bottles containing this serum, *i.e.* having the *same number* on them, which are still on the market are called in.

In Germany at the present time diphtheria curative serum is prepared in four different places, but it has to conform to official tests before it can be sold. More serum is prepared at Höchst-a.-M. than anywhere else, and four different forms are made at Höchst.

No. 0 contains 0·8 cc. of 250 times normal serum = 200 immunising units. An immunising dose.

No. 1 contains 2·4 cc. of 250 times normal serum = 600 units. A single curative dose for cases in which treatment can be commenced on the first appearance of symptoms.

No. 2 contains 4 cc. of 250 times normal serum = 1000 units. A double curative dose, sufficient for most cases of diphtheria when treatment begins not later than the commencement of the third day.

No. 3 contains 6 cc. of 250 times normal serum = 1500 units. A triple curative dose for very severe and advanced cases. The works at Höchst also manufacture a serum twice as strong as the preceding, *i.e.* 500 times normal. It too is obtainable in four forms, which have each, therefore, twice as many units as the preceding preparations.

The curative serum keeps very well, as a rule, for at least one year, if it be kept in a cool place and protected from light and frost. Still more durable than the fluid preparation is the dry one prepared at the works at Höchst-a.-M. by careful evaporation, and which is placed on the market under State control. It is a pure evaporated serum to which no antiseptic or other substance has been added. The preparation is in the form of fine yellowish scales or a yellowish-white powder, and before being used must be dissolved in ten parts of distilled water, which takes some time. To meet official requirements 1 gramme of the dry preparation must contain at least 5000 units.

The standardisation of tetanus curative serum is essentially the same as in the case of diphtheria. Behring (and also Knorr) employed a normal tetanus antitoxin (Tet. AN), which must again be standardised by means of a tetanus toxin the nature of which is constant, and such a toxin can only be obtained by the addition of ammonium sulphate and the drying of the precipitate so obtained (Buchner). For standardisation in the *Institut f. experimentelle Therapie* at Frankfurt-a.-M. there is now used, not a dry toxin, but a ten times normal serum, which has been evaporated to dryness, for this preparation remains unchanged for years if attention be paid to the conditions under which it is kept, as above described. All tetanus serum made in Germany is subjected to official tests, as is diphtheria serum. Tetanus serum is prepared in Germany at the works at Höchst-a.-M.; the serum was formerly prepared both in fluid and solid form, but now only in the former. Each bottle contains 25 cc. of ten times Tet. AN, equal to 250 antitoxic units, suited for treating men and animals suffering from tetanus. Tetanus serum, which is protected from bacteria by the addition of 0·25 per cent *metakresol*, can be preserved for a long time, at least one year, if kept in a cool, dark place and preserved from frost.

The firm of Merck (Darmstadt) in addition supplies Germany with Tizzoni's tetanus serum, which is a dry preparation. Köhler's statistics show that it acts just as well as Behring's preparation, provided it can be injected early enough, repeatedly and freely. Before use, the dry preparation is to be dissolved in ten times the quantity of sterile fluid, and 1 cc. of the solution then contains 80,000 immunisation units, *i.e.* is able to completely neutralise 80,000 toxic units. Tizzoni's toxic unit represents the minimum quantity of filtered tetanus bouillon which can kill 1 kilo. living weight of rabbit in five to six days.

3. *The Cause of the Antagonism of Antitoxin to Toxin.*—Now there are three conceivable possibilities. Behring at first thought that there was a destruction of poison by means of the antitoxin. But if this were the case, a given quantity of antitoxin, if remaining longer in contact, would be able to gradually destroy more toxin; a mixture of antitoxin and toxin, which had at first a toxic action, would after a time have a neutral action, and would finally contain surplus antitoxin. This, however, is not the case (Roux and Vaillard, Buchner). It was also shown that a mixture of tetanus toxin and antitoxin which is almost neutral for a mouse can, when absolutely the same dose is injected, cause fatal tetanus in the guinea-pig, which is about eighteen times as heavy (Buchner). Hence in a mixture which is neutral for a mouse the poison is not completely destroyed.

This fact, however, harmonises with a second theory propounded by Roux, according to which the action of antitoxin is to be regarded as an indirect one—the antitoxin acting on the living elements of the organism and immunising them to the toxin. Roux based this view on the fact that the action of antitoxin is completely different in a healthy organism, and in one previously weakened, a fact which could not be explained by the supposition of a direct destructive action of antitoxin on toxin. In support of Roux's theory is the fact that antitoxic action is not in all cases strictly specific. For example, the blood-serum of animals which have been treated with tetanus toxin is antitoxic, not only to that toxin, but also to snake venom. The blood-serum of rabbits which have been immunised against rabies is not protective against rabies, but is markedly antitoxic to snake venom (Roux and Calmette). Similarly, the serum of animals immunised against snake venom is also antitoxic to scorpion poison (Calmette, Metschnikoff), and the immune sera obtained by means of ricin, abrin, etc., yield similar results (Ehrlich).

In spite of these facts this second theory, according to which antitoxin and toxin are *physiological antagonists*, cannot be regarded as correct. There is more probably a direct combination of antitoxin and specific toxin, whereby the action of the latter on the organism is arrested, as was first shown by Ehrlich and then by Knorr. But here too it is necessary to determine more definitely the nature of this combination which Ehrlich regards as a simple chemical one like that between acid and base. Recent investigations carried out by Ehrlich with ricin and antiricin, by H. Kossel, Camus and Gley, and Kanthack, prove beyond doubt that antitoxin and toxin directly affect and combine with each other. Possibly, however, there is not a simple chemical combination, but a sort of absorptive action, such as we see in connection with staining agents. This latter supposition is as a matter of fact by no means improbable, for it is only thus that a series of facts ascertained by A. Knorr can be explained. There is no doubt that toxin and antitoxin generally combine in proportionate amounts, so that if one unit of toxin be neutralised by a given quantity of antitoxin, one hundred times as much antitoxin is able to combine with one hundred units of toxin. Yet there are deviations from this rule. One striking fact is that a concentrated mixture of toxin and antitoxin differs from a dilute mixture in regard to neutralisation. For instance, Knorr, using the same toxin and antitoxin in each case, obtained the following results:—

Units toxin.		Units antitoxin.		
250,000	+	250,000	=	0
2,500	+	2,500	=	Mild tetanus
25	+	25	=	Mild tetanus

Again, the length of time the toxin and antitoxin have been in contact before being injected is of importance in determining their combination; for example, a mixture of 2500 units toxin + 2500 units antitoxin immediately after being prepared was injected into an animal, and death from tetanus occurred three and a half days later. The same mixture if kept for two hours caused only slight disease, and after being preserved for twenty-four hours it became inactive. Such combinations vary very greatly according to the nature of the poison in question. The subject has only become comprehensible since Ehrlich showed how greatly the constitution of various preparations of one and the same poison may differ, according to the amount of toxones, toxoids, etc., they contain, *i.e.* the amount of modified toxins which are non-poisonous, and yet which combine with antitoxin. These differences in the various toxic preparations, therefore, do not help to solve the problem. Now, if there were merely a chemical combination of toxin and antitoxin, it would necessarily follow, when dealing with one and the same solution of toxin and antitoxin, that the degree of concentration would be of little importance. In point of fact, however, this is not so. Knorr, for example, working with his standard toxin and standard antitoxin, obtained the following results:—

Units toxin.	Units antitoxin.	
250,000	+ 250,000	= 0
	+ 225,000	= Mild disease.
	+ 200,000	= Death in 4 to 9 days.
	+ 150,000	= Death in $2\frac{1}{4}$ to 4 days.
	+ 125,000	= Death in $1\frac{1}{2}$ to $2\frac{1}{2}$ days.
	+ 100,000	= Death without delay.

Thus in the fourth experiment 100,000 units of toxin were reckoned as being in excess. *Their physiological action, however, is merely equivalent to that of* 15 *units of free toxin,* and to compensate the toxic effect of mixture No. 4, and to render it neutral again, there are not 15 units of antitoxin but 100,000 units required.

The inference is that in a concentrated solution a unit of antitoxin may sometimes combine loosely with more than one unit of toxin, so that the latter's toxic action is greatly diminished. This too is the real cause of the results experimentally obtained by Buchner, who, as already noted, found that a mixture of tetanus antitoxin and toxin which is almost neutral for a mouse, can produce fatal effects in guinea-pigs. The loosely combined portion of the toxin has an action which varies in degree according to the specific susceptibility to toxin of the different animal species. The exact nature of the combination between antitoxin and toxin is still unknown, but the fact that there is a combination cannot be doubted. The antagonistic effect of antitoxin on toxin is due to this combination, which is therefore one of the most important facts in the realm of immunity.

Another no less important question is that of the *nature and origin* of antitoxins. Behring considers them to be products of the animal body, formed in consequence of the feverish reaction attending injection of toxic filtrates. This, however, is not invariably so, for, according to Metschnikoff, the crocodile (Alligator Mississippiensis) is distinguished from all other animals by the rapidity in the formation of antitoxin, *although there is no feverish reaction.* Twenty-four hours after the injection of tetanus toxin and six days after the introduction of cholera toxin in solution, the alligator's blood becomes antitoxic to these poisons to which it is very insusceptible. Yet, when full-grown alligators are contrasted with young crocodiles and turtles, it is remarkable that both the latter form antitoxins very slowly if at all, and are yet very insusceptible to tetanus toxin, and can stand enormous doses of it.

Ehrlich has recently furnished, by means of his "*side-chain theory,*" a definite and in many respects a very instructive explanation. According to this theory the general action of toxins is as follows:—Each molecule of toxin possesses two different chemical groups; one is "haptophorous," and by means of this the toxin becomes firmly bound to any cell protoplasm of the organism for which it has a chemical affinity, and a "toxophorous" group by which disturbance is then induced in the protoplasm in question. The toxin does not become bound to the "functional centre" of the cell protoplasm, but to a side chain of the latter. Hence the name of the theory. If the action of the toxophorous group of toxin molecules causes disturbance in the cell protoplasm, and if certain side-chains are consequently destroyed, the latter, it is said, are re-formed by the regenerative efforts of the organism. If this process be of considerable intensity, there may be excessive production of side-chains, the excess is transferred to the blood, and possessing, as already mentioned, a chemical affinity for the haptophorous group of the given specific toxin, acts as antitoxin. This ingenious theory explains in a relatively simple way the remarkable fact that the organism responds to the introduction of toxin by the formation of a specific antitoxin. The supposition that the action of toxin in the body is dependent on the chemical affinity of toxin for definite cell protoplasm is undoubtedly correct. The theory, however, is open to hostile criticism (*vide infra*).

Another theory, held by Buchner and Metschnikoff, is that antitoxins represent in general non-poisonous modifications of toxins, which are elaborated by certain cellular elements of the body and transferred to the blood. Space forbids a detailed account of the facts on which this view is founded, but it may be said that this view harmonises better than does the side-chain theory with recent observations which indicate that the organism reacts with the formation of antitoxin, not only after the introduction of bacterial toxins, but that it reacts similarly on the introduction of the red blood corpuscles of any other species of animal, forming a specific anti-substance which passes into the blood; the same occurs on the introduction of foreign epithelium, leucocytes, spermatozoa, etc. The elaboration of anti-substances must therefore be regarded as a universal mode of reaction against foreign cells and their products, and not merely against toxins.

4. *Transference of Antitoxin by Inheritance and Suckling.* (*Milk.*)—Ehrlich showed that in mice rendered tolerant of ricin or abrin the immunity is transmitted to the offspring from the mother, but not from the father, and he found that this tolerance of poison is only in part transferred from the mother's serum, but is mainly due to a subsequent transference of antitoxins by means of suckling. He proved this fact by the "exchange or wet-nurse experiment," in which the newly-born offspring of a highly-immunised mouse and that of a control mouse were respectively suckled by the different mothers. The offspring of the highly-immunised mouse, on being suckled by a normal control animal, possessed only a very slight degree of immunity after twenty-one days, in consequence of the excretion of the antitoxins originally inherited from the mother, whilst the immunised

mother was able to transmit the antitoxin by her milk, and a marked and progressive immunity was thus conferred to the offspring of the control animal. The transference of immunity or tolerance of poison to the offspring by means of the milk was subsequently demonstrated in tetanus (Ehrlich), and it was shown that the subcutaneous injection of the milk of goats which have been immunised against tetanus confers protection from tetanus (Brieger and Ehrlich). Antitoxin has been successfully precipitated from milk and prepared in a dry concentrated form. The antitoxins contained in milk are absorbed from the intestinal canal without undergoing change mainly because of their marked stability. Ehrlich shows that the maternal milk may also be serviceable for the transference of antitoxin to the human infant, and indicates that it is possibly owing to this fact that a number of infectious diseases (mumps, scarlatina, measles) so seldom occur during the first year of life.

5. *Employment of Immune Sera for Immunisation and Curative Purposes.* (*Prophylactic Employment and Serum Therapeutics.*)—Two forms of immune serum can be differentiated: the antitoxic serum, and the serum the action of which is only anti-bacterial or bactericidal. Results of real value have hitherto only been obtained with antitoxic serum, but brief mention must be made of the results gained by the use of bactericidal sera. In diphtheria and tetanus more especially it has long since been proved experimentally that a cure may be effected by antitoxic serum even after the disease has commenced (Behring, Kitasato, Wernicke, Knorr), the explanation of the curative effect being the above-mentioned combination of antitoxin and toxin. But a relatively large amount of antitoxin is required if treatment is to be successful, and the longer the time elapsed since the commencement of the illness the more antitoxin is necessary. In the late stages of the disease the intoxication is so great that large doses of antitoxin are ineffectual. Under such circumstances an antitoxin of the highest possible concentration is of great value, and it has often been successfully prepared by precipitating the antitoxin from the serum and subsequently making a concentrated solution. Another method of gaining the same end is to freeze and then thaw the serum, thus separating water and proteid, a process which may be promoted by centrifugalisation.

Diphtheria.—Antitoxic diphtheria serum is but seldom used for immunisation. The usual dose is 200 units, and if danger of infection persists the inoculation is to be repeated about fourteen days later, because of the rapid excretion of the antitoxin from the body. The use of larger doses of serum is not so suitable, because the more concentrated the antitoxin in the blood the more rapid is the excretion. It is difficult to furnish absolute proof of the protection man derives from these inoculations; to do so would require many statistics. The results gained by Löhr and Slawyk in the children's clinic at the Berlin Charité are interesting. By immunising all the children, including those newly admitted, they succeeded in checking the spread of diphtheria in the clinic, as could not be done otherwise, whilst mere immunisation of those children in beds adjoining that of a diphtheria case had failed to do so. The immunising dose was usually 200 units, which was borne without risk.

The *therapeutic* application of diphtheria serum is based on the experimental studies of Behring and Wernicke (1892) on the recovery of guinea-pigs after subcutaneous inoculation with diphtheria bacilli in such doses that the animals would have died in three or four days had they not been treated. If treatment were commenced immediately after infection, $1\frac{1}{2}$ to 2 immunising doses were required to effect a cure; eight hours after infection, 3 immunising doses; and twenty-four to thirty-six hours after infection, 8 immunising doses were requisite. After small doses of diphtheria bacilli, when the disease in the guinea-pig runs a slower course, the curative action can also be recognised in the local changes at the seat of injection, for the serous exudation which has been formed quickly disappears. If infection has occurred some considerable time previously, so that the disease is already advanced, large doses of antitoxin cannot save the animal. The disturbances, which are already in existence in the various cells of the body, can no longer be influenced by the antitoxin. The action of diphtheria serum can also be experimentally shown in the case of a true superficial diphtheria produced by inoculation of diphtheria bacilli on the vagina, trachea, or other mucous membranes of animals. The false membrane so formed becomes detached as the animal recovers (Roux and Martin). The action of the serum is only antitoxic, not anti-bacterial; but as the diphtheria bacilli do not spread into the body from the seat of inoculation, the organism, after the use of curative serum, gains time to gradually overcome the bacilli by means of its natural protective powers.

For use in medical practice the rule is to inject large doses as soon as possible, giving at least 1000 units, in severe and urgent cases 1500 to 3000 units, or even more. The antitoxin must be regarded as absolutely innocuous; any by-effects, such as skin eruptions or pains in the limbs, which sometimes occur, are due solely to the serum, normal horse's serum being able to cause similar symptoms. The injections are made with a sterilised syringe under the skin of the thigh, or between the shoulders, with strict attention to asepsis. The serum must be clear or somewhat opalescent, it should

not show cloudiness. Statistical proof of the curative action of the serum has been furnished from all sides; for example, Roux's experience is that since the introduction of curative serum the mortality has fallen from 50 per cent to 26 per cent. The decrease of mortality according to Heubner is from 44·3 to 21·1 per cent, according to Baginsky from 48·4 to 15·6, according to Ranke from 57 to 18·8, and according to Monti from 34 to 22 per cent. The joint inquiry of the *Deutsche medicinische Wochenschrift* showed that in 5833 cases treated with serum the mortality was 9·6 per cent, whilst it was 14·7 per cent in 4479 cases not treated with serum. Statistics also prove that the sooner treatment is begun the more favourable are the results. The following are the results published by the German Imperial Board of Health, the investigations dealing with 204 hospitals in Germany for the period from April 1895 till March 1896:—

Total number of cases	9581	
Total mortality	15·5	per cent
Mortality when treated on 1st day	6·6	,,
,, ,, ,, 2nd ,,	8·3	,,
,, ,, ,, 3rd ,,	12·9	,,
,, ,, ,, 4th ,,	17·0	,,
,, ,, ,, 5th ,,	23·2	,,
,, ,, after 5th ,,	26·9	,,

Behring's experience is that those cases of diphtheria in which treatment with a single curative dose (600 units) is begun within forty-eight hours of the commencement of the disease will have a mortality of less than 5 per cent.

Many doctors, moreover, maintain that the clinical course of disease is favourably influenced by the curative serum. There are those, however, who hold a contrary opinion, which is perhaps due to the fact that in diphtheria there is often a mixed infection with streptococci, and that the latter are not influenced by the serum. But the majority of observations indicate that it is more especially in laryngeal diphtheria that injections of serum induce the most favourable effect, the signs of stenosis usually disappearing rapidly after the injections. It cannot be denied that post-diphtheritic paralyses are as numerous since the introduction of serum treatment, or that they have become more frequent still; but this is possibly due to the fact that a greater number of severe cases recover now than formerly. It is often said that curative serum causes albuminuria. According to the statistics of the German Imperial Board of Health, two-thirds of the cases treated with serum had no albuminuria, although it is by no means of rare occurrence in diphtheria. Unpleasant by-effects, such as skin rashes of an urticarial character, joint pains and swellings, can doubtless be caused by the diphtheria curative serum. The cause thereof is not the antitoxin, but the serum, which even when normal can induce such conditions in persons predisposed thereto. Such effects are in no way dangerous, but it would be well if the antitoxin could be separated from the constituents of the serum and prepared in a pure form.

Tetanus.—The prophylactic use of serum is of greater importance in tetanus than in diphtheria. Immunisation against tetanus was carried out by Nocard (1895-97) in 2373 horses and 332 other large animals. None of these succumbed, whilst during a similar period of time and under the same conditions there had previously been 191 cases of tetanus in horses and 68 cases in other animals.

Immunisation, therefore, for which small doses costing but little are sufficient, is advisable previous to any operations on the horse when cleanliness is a matter of difficulty, and also after such wounds as are known to be often followed by tetanus. In man, too, tetanus serum as a prophylactic agent is very useful after severe contused wounds contaminated with soil, dust, manure, etc. The duration of the immunity conferred is comparatively short except when horses are inoculated with tetanus serum obtained from horses.

Behring and Knorr, injecting tetanus toxin into animals, have experimentally investigated the possibility of curing tetanus with serum. If only the minimum lethal dose be given it may be possible to save the animal by the introduction of serum even a considerable time afterwards. If, however, the intoxication be induced by a larger quantity of toxin—for example, 100 times the minimum lethal dose—the amount of antitoxin subsequently required becomes much greater. Even a quarter of an hour after the injection of toxin it is necessary, in order to effect a cure, to use 100 times as much antitoxin as would have sufficed to neutralise the same quantity of toxin had they been mixed *in vitro* prior to injection; and very soon, long before any tetanic symptoms appear, it becomes impossible to save the animal. It is much easier to cure animals after they have been infected with tetanic spores, as occurs in natural infection, than when they have been poisoned with prepared toxin (Kitasato, Knorr), and such experiments prove that the antitoxin may really effect a cure even though symptoms of the disease have appeared. The difficulty of curing an advanced case of tetanus by means of serum is probably due to the fact that the toxin is soon absorbed by and bound to the tissues of the body, and the neutralising effect of the antitoxin thus inhibited. It has been shown in rabbits that to prevent the onset of tetanus a dose of serum, if given twenty-four hours after the injection of toxin, must be twenty-four times as great as that which suffices when given at the same time as the toxin (Dönitz). This indicates that even in the first few hours the greater part of the toxin has combined with the tissues of the body, for otherwise subsequent neutralisation in the blood would be possible by

means of the antitoxin. In spite of this combination a sufficient amount of serum may even yet effect a cure, hence we must assume that antitoxin when present in abundance is able to withdraw the loosely combined toxin from the tissues, or to neutralise it while still there.

The results hitherto gained in large animals, and especially in horses, are indeed not very favourable, but would undoubtedly be better were treatment begun sooner and larger doses given. In man the difficulties in the way of successful treatment of tetanus with serum are still greater, and there is no statistical proof that the results have improved since antitoxic serum was introduced. This is obviously due to the fact that by the time the first tetanic symptoms appear, the toxin has already become combined in the nervous system, and that the disturbances there can no longer be influenced by the antitoxin. The great importance of the prophylactic use of tetanus serum must therefore be again emphasised, for in this case the antitoxin present in the body may be confidently expected to neutralise the toxin on its first appearance in the blood, and *before* it has combined with the tissue elements. The attempts to render the toxin more active by means of intra-venous injection, or by direct introduction beneath the dura mater (Roux), have hitherto not yielded results of practical value.

Plague.—Plague serum is prepared by treating horses with increasing doses of living or killed plague cultures, and usually by means of intravenous injections (Yersin, Roux, Wladimiroff). Each injection causes fever and signs of a general reaction. A serum is finally obtained which in even somewhat small doses can protect other animals against infection with plague. At the Pasteur Institute in Paris the serum is standardised by estimating the smallest dose which can protect mice against infection twenty-four hours subsequently with the minimum lethal dose of plague bacilli. Yersin in 1897 obtained favourable results with such a plague serum, for of 500 inoculated individuals in a plague-stricken district only 5 sickened and 2 died. In 2 of these 5 cases the disease occurred so soon after the inoculation that infection had probably already occurred, and they should not be counted. The symptoms in the other 3 cases ensued twelve to forty-two days after inoculation, *i.e.* probably after the protective effect had expired, for we may conclude from animal experiments that the protection conferred only lasts ten to fourteen days. The experimental researches of the German Plague Commission on the immunising value of Yersin's plague serum demonstrated that by means of 10 cc. monkeys can generally be immunised against several times the minimum lethal dose. There is, however, a form of grey monkey which has a special susceptibility to plague, and in which the protective action of the serum is of no avail, and it therefore appears doubtful whether effective protection can be obtained for the human subject.

The above-mentioned short duration of the protection conferred by plague serum is, however, a practical drawback, which does not exist in the active immunisation with killed plague cultures. In the most urgent cases a combination of active and passive immunisation—injecting killed cultures and plague serum together—would perhaps be advisable.

On the therapeutic application of plague serum, reports were given by Yersin from India (1896 and 1897), which, however, require further confirmation. The German Plague Commission had the opportunity in Bombay of studying the results of serum treatment in a series of 26 individuals, whose ages varied from $1\frac{1}{2}$ to 60 years. They were all recent cases of simple bubonic plague, in whom the prognosis would in any case have been favourable, so that although the mortality in these cases was equal to 50 per cent, and therefore appears more favourable than the usual average mortality (80 per cent) from plague, it is no proof that the serum had a curative effect.

Both the German and the Russian Plague Commissions investigated the curative effect of the serum in animals. In one breed of monkeys, which are not very susceptible to plague, a cure was effected by means of 10 cc. of serum even six and twelve hours after infection with a lethal dose of plague cultures. If the administration of the serum were further delayed the result was uncertain or negative. The results will undoubtedly be better if a more potent plague serum can be prepared. The serum is not, or only to a slight degree, antitoxic; its action is mainly a specific bactericidal one, just as in animals treated with cholera and typhoid bacteria. The condition in plague is more a bacterial infection than an intoxication, as is also the case in anthrax, for example. Roux, however, believes that the antitoxic action of plague serum is dependent on its mode of preparation, and that the serum contains most antitoxin when obtained by the intravenous injection of living plague bacilli.

Cholera.—In cholera, as in plague, few definite results of practical value have been obtained. It is indeed easy to obtain a specific bactericidal serum by treating animals with cholera vibrios. But the value of such sera for immunisation appears to be slight; large doses would have to be used, and in spite of them the protection would be of short duration. Behring and Ransom, Roux and Taurelli-Salimbeni attempted to isolate a toxin from cholera vibrios, and to obtain an antitoxic cholera serum by treating horses with this toxin. It is claimed that this serum protects not merely against cholera intoxication, but also against the living cholera virus. No use has as yet been made of

this serum on the human subject. In man, therefore, active immunisation by killed cholera cultures appears as yet to be the only practical method of proved value.

The *therapeutic* value of cholera serum is naturally still more doubtful, especially when the rapid course of the disease is borne in mind. Attempts to treat cholera patients with the blood serum of those who had recovered from the disease have been very indecisive.

Typhoid Fever.—By treatment with killed cultures (R. Pfeiffer), a serum is obtained which is specifically bactericidal in a moderate degree, but not antitoxic, and therefore unsuited for therapeutic purposes in man. For intoxication is the main feature in typhoid, and an antitoxic serum would therefore be required. Pfeiffer's results have been corroborated by many writers. Beumer and Peiper obtained a serum from sheep by a method essentially the same as Pfeiffer's, but it had no curative action in typhoid. Apparently good results were obtained by Klemperer and Levy, who got the serum from dogs, but the typhoid cases in which it was employed were mild, so that the observations are not decisive.

Pneumonia.—Attempts to obtain an immune serum and to employ it in pneumococcal infection in animals were formerly made. G. and F. Klemperer were the first to employ the serum of rabbits, which had been treated with pneumococci, in pneumonia in the human subject, having previously proved that such serum can effect a cure in pneumococcal infection in rabbits. They sought to prove the etiological identity of these two diseases by showing that the serum of persons convalescing from pneumonia can also bring about recovery from pneumococcal infection of rabbits. An injection of 4-6 cc. of the serum appeared to influence the course of pneumonias favourably, in that the temperature often fell and remained down. Observations were, however, only made on six cases, so that no definite opinion can be given. The action of the serum is probably bactericidal and not antitoxic (Issaëff and Bonome).

Streptococcal Infections.—Behring, Knorr, and others had worked at immunisation against streptococci, but without definite success. In 1895 Marmoreck claimed to have obtained an antistreptococcus serum from rabbits, and afterwards from horses. Favourable reports as to its value for the human subject were soon published in France. For immunisation, horses are subcutaneously inoculated with gradually increasing doses of cultures of streptococci, which are highly virulent for rabbits. Each injection is given only after the complete disappearance of the signs of reaction, which are very intense. The serum of such animals is then claimed to exert a protective and curative action in streptococcal infections. This was experimentally confirmed by Bordet, but others (Aronson, Petruschky) obtained absolutely negative results. The apparently favourable results attained with this serum in erysipelas and puerperal fever are too scanty, and the use of the serum does not sufficiently explain the direct change induced in the temperature, etc.

It was afterwards asserted by Denys and others that a given streptococcus can induce immunity and furnish a protective serum only against streptococci of the same stock, and that a number of streptococci of different stocks must therefore be simultaneously made use of when immunising. But it was also shown that streptococci of different stocks which are virulent only for animals may be quite unsuitable for the production of a serum which is to be employed in man. Hence Tavel for the immunisation of horses only employs such streptococci as have been obtained directly from the human subject. There is but little known of the clinical results obtained by such serum. But on the whole it is improbable that a potent antistreptococcus serum will be obtained, because the serum of persons who have recovered from a streptococcal infection has no power of immunising against the same streptococcus, and again because streptococcal infection in man confers no protection, or even predisposes to a subsequent attack, as in erysipelas of the face, for example. The apparent benefit from the use of such serum in many clinical cases is possibly due to an increase of natural resistance induced by hyperleucocytosis.

Staphylococcal Infections.—Animals can be rendered immune to staphylococci by giving them increasing injections of cultures of staphylococci. Viguerat rendered goats highly immune by this method, and then used their serum for therapeutic purposes in staphylococcal infections in man. But the results recorded cannot be regarded as proof of a curative action. Subsequent investigations by Petersen have indeed confirmed the fact that animals can be immunised against staphylococci, but the value of the immune sera obtainable is too slight to admit of the serum being of practical value for the human subject. According to Petersen these sera are anti-bacterial and not antitoxic.

Intoxication with Snake Venom.—According to Calmette, the various varieties of snakes have different venoms, and in a given snake the virulence varies according to the length of time the snake has fasted, becoming gradually and steadily greater. Rabbits, guinea-pigs, donkeys, and horses can gradually be rendered immune to what is otherwise a lethal dose of venom by treating them at first with small and later with increasing doses of venom. Their serum then possesses antitoxic and curative powers, both of which, according to Calmette, are tested on rabbits. A rabbit is first given 2 cc. of serum, and five minutes later the minimum lethal dose of snake venom is injected intravenously. With-

out the serum the animal would die in 15 to 30 minutes; if the serum be potent the animal remains healthy. To test the curative value, an analogous method is employed. The lethal dose is injected into a vein of a rabbit, and five minutes later, when the venom is commencing to take effect, 4 cc. of serum are given intravenously. The animal should then recover in a short time. All the serum prepared by Calmette has to conform to these two tests.

It is important to know whether the venoms of the various varieties of snakes are essentially identical. Cunningham differentiates two forms, *i.e. blood poisons*, such as the venom of cobra capella, and *nerve poisons*. Calmette holds a contrary opinion, and refers to the fact that his serum has been used with excellent results for the bites of quite a number of different snakes. His serum is even claimed to be antitoxic to scorpion poison (a point corroborated by Metschnikoff), although the latter is undoubtedly different from snake venom.

Owing to this fact and to others previously mentioned, we may conclude that the antitoxic action is not so markedly specific in the case of these venoms as in the bacterial poisons. The former also differ from the latter in being more resistant to high temperatures, undergoing no change at 80° to 90°. Calmette's assertion, that the serum of an animal which is rendered highly immune to a given snake venom also confers protection against the venoms of other varieties of snakes, is accordingly by no means improbable.

Calmette's results have been in the main confirmed by Fraser, who succeeded in obtaining a very potent anti-venom serum from the horse. According to Fraser, 200 to 300 cc. are required to save a man after being bitten. The serum has hitherto not been sufficiently used in India to permit of definite conclusions being drawn. Fraser states that the bile of venomous snakes may also be antitoxic. By precipitating with alcohol, he obtained an antitoxin of high curative value, which, even when given thirty minutes after a lethal dose of cobra poison, was able to save the animal, the proportionate weight of antitoxin employed to body-weight being 0·01-0·07 : 1000. Calmette regards this phenomenon as not being due to the action of an antitoxin, but considers the bile substance as an unusually active stimulant, yet such an explanation does not diminish the practical significance of the fact.

In every form of snake bite Calmette recommends the employment not only of antitoxin, but also of chloride of lime solution, freshly prepared, to be used as a wash and also subcutaneously injected, after a firm ligature has been placed round the limb; 20 to 30 cc. of antitoxic serum are to be injected under the skin of the abdomen.

Tuberculosis.—Behring has striven for many years to obtain an antitoxic serum for tuberculosis. His aim was to first prepare a potent toxin from tubercle bacilli and then to immunise animals therewith. He succeeded in preparing a toxin the toxic activity of which is considerably greater than that of Koch's tuberculin. Tuberculous cattle were cured by gradually increasing doses of Behring's toxin, and simultaneously rendered immune, so that they were finally able to withstand a dose of toxin lethal to healthy cattle. But the serum of such animals possessed only a slight protective power against Koch's tuberculin. In regard to the production of a tuberculosis antitoxic serum, Ransom hoped to get better results from birds, but none of practical value have been recorded.

The employment of birds was mainly suggested by the fact that the mere use of the serum of horses and cattle, not only of those which have been inoculated, causes fever in phthisical patients, which is not the case with the serum of most birds.

At an earlier date Maragliano stated that he had obtained an antitoxic serum by treating the larger animals, and horses more particularly, with tuberculin toxin. The simultaneous injection of tuberculin and this serum is said to cause neither local nor general reaction. This serum has been much used by Maragliano and other Italian physicians, but there is no definite proof of it having been employed with success. It is on the whole very doubtful whether a true antitoxic serum can be prepared in the case of tuberculosis, for in this disease the toxic action is not very pronounced, and the experience of most men is that recovery does not confer immunity.

Meat Poisoning.—The B. botulinus is frequently an etiological factor, and its toxins are somewhat analogous to those of diphtheria and tetanus. After treating animals with filtrates of bouillon cultures of B. botulinus, they may furnish an antitoxic serum which has both a prophylactic and curative action (Kempner).

Swine Erysipelas.—Lorenz's method of protective inoculation is the most successful and most frequently used in Germany. It is based on a judicious combination of active and passive immunisation. The possibility of immunising against swine erysipelas by either method had previously been pointed out by Emmerich. Yet passive antitoxic immunisation alone has the disadvantage of conferring a protection of too short duration, whilst simple active immunisation with living cultures implies the risk of inducing serious disease. What Lorenz aimed at was, firstly, to render the animals somewhat immune by injections of serum, and then a few days later to inject $\frac{1}{4}$-1 cc. of a living bouillon culture. Protection is thus conferred and lasts for about five months. If this be not considered sufficient, another injection of 0·5-2 cc. of living culture must be made about a fortnight later, when the

resulting protection lasts for about one year. Before the end of that time further injections may be given and the protection indefinitely prolonged. Lorenz has of late obtained his serum from horses, and he treats it so that it shall keep.

Of 4540 swine immunised by this procedure in Germany in 1896, only two died, and that too seven months after treatment. According to Voges and Schütz this procedure is best adapted for the finer breeds of swine, whilst Pasteur's method, as already mentioned, is more suitable for the ordinary breeds.

Cattle Plague (*Rinderpest*).—The investigations of R. Koch, Kolle, and Turner show that there are two main forms of protective inoculation for cattle plague, the virus of which is still unknown. The one method is the use of the bile of animals which have died of cattle plague. The injection of 10 cc. of such bile confers, about ten days later, an immunity which last for several weeks, and which according to Koch and Kolle is an *active* one, the bile containing the attenuated virus. Compulsory inoculation with bile was attended, in Basutoland for example, with the following results: of 100,000 animals 70,000 remained alive, whilst all the uninoculated herds died. Kohlstock modified and improved this method; subsequent to inoculation with bile he injects 1 cc. of virulent cattle plague blood.

As the bile of 3-7 oxen is required for the immunisation of 100 cattle, the *combined* use of serum and cattle plague blood is as a rule better than the "bile method." Cattle which have recovered from the disease are given increasing injections of cattle plague blood. They eventually become highly immune, and furnish a very potent serum which may even effect a cure after the disease had commenced. 15-40 cc. of such serum are injected into one flank, 1 cc. of virulent cattle plague blood being injected at the same time into the other flank (simultaneous method). The results appear to be very satisfactory; of 9007 inoculated animals only 178 (1·4 per cent) died of cattle plague, and moreover the immunity thus obtained lasts for some months. This latter method is not only the more effective, but also the cheaper of the two, since a highly immunised animal can furnish serum for 300 others. Yet the "bile method" is of service when a herd is attacked with the disease, and there is then a sufficient supply of the vaccine.

Conclusion.—The latest discoveries in connection with immunity are characterised by the *specific hæmolytic* actions which Bordet was the first to investigate in detail. If a definite quantity of rabbit's blood be introduced into the peritoneum of a guinea-pig, the red blood corpuscles of the rabbit are somewhat slowly dissolved and eventually disappear completely. If, however, the same animal be given such injections repeatedly, its serum requires a *specific hæmolytic* action on the rabbit's red blood corpuscles, so that they are much more rapidly destroyed than they would be in the serum of a normal guinea-pig. But the increased hæmolytic power of the serum has reference only to the red blood corpuscles of a rabbit, and not to those of any other animal. The condition is therefore analogous to the specific bactericidal actions. In a similar manner the serum of a rabbit may acquire a specific hæmolytic action on the red blood corpuscles of the ox or the dog, after being treated with the blood of the ox or the dog respectively. The same rule probably holds good for the red blood corpuscles of all mammals in relation to all the different species of mammals (*vide infra*).

Analogous conditions have been demonstrated for other forms of animal cells (ciliated epithelium, leucocytes, spermatozoa) by v. Dungern, Metschnikoff, and others, for after treating an animal with such cells its serum acquires a specific destructive power on similar cells. Such specific treatment, which incites the elaboration of specific protective substances in the body, can therefore be carried out in an indefinite number of instances.

Bordet proved that specific hæmolytic action is similar to specific bactericidal action in being due to the *combined action of two different substances*, on the one hand that of a specific anti-substance which is preserved at 65° C., and on the other that of the non-specific alexin which effects the dissolution of the hæmoglobin. The process in detail is that the specific anti-substance is attracted and bound to the blood corpuscles in question (Ehrlich and Morgenroth), which thereby become predisposed to the solvent influence of the alexin. The analogy to the specific bactericidal action is therefore complete; in the latter instance the specific anti-substance (agglutinin) which is preserved at 65° C. becomes bound to the specific bacteria (Gruber, Hahn, and Trommsdorff), which are thereby rendered susceptible to the action of the alexins.

But we also know that the antitoxins—the specific substances of tetanus, diphtheria and other sera, which are preserved at 65° C.—neutralise the specific toxins by forming a sort of loose combination with them, and that here at any rate the action is in no way due to the alexins, because there are in this instance no foreign living cells.

In consequence of all this, the essence in all cases of *specific* immunity consists in the fact that *the organism is protected by the formation of a specific definite anti-substance or antitoxin which is preserved at* 65°, *and which has a peculiar attraction for any foreign invader* (toxin, bacteria, red blood corpuscles, etc.), *and that what is thereby effected is a neutralisation* (of poisons), *or an alteration permitting the protective action of the normal alexins to be displayed* (in the

specific bactericidal, specific hæmolytic actions, etc.).

The condition in which the organism because of prolonged administration of poison becomes accustomed to it, or as is commonly said becomes immune, is not in every case due to the formation of antitoxin. According to recent researches by Edw. Faust the increased tolerance of morphia is not due to antitoxin formation, but to an increased capability of the body of destroying morphia. *True immunisation with the formation of antitoxin is only possible in the case of substances without definite chemical constitution* (proteids or their allies), and not in the case of simpler molecules.

Lastly, we have once more to note that the phenomena of immunity are *in part to be included in the realm of "specific immunity,"* and in part in that of "natural immunity" for which we have employed the term "natural resistance." "Specific immunity" and "natural resistance" are in theory totally distinct conditions, but in practice they very frequently, almost always, act in combination in one and the same individual in warding off the infective agent. An infective process cannot be correctly understood without taking into consideration both forms of immunity and their variations. The difference between the two forms of immunity is best characterised by their active substances: the alexins of natural resistance are destroyed at 60°, vary according to the animal species forming them, and are not attracted to the toxins, bacteria, etc. The antitoxins and anti-substances of specific immunity do not derive their character from the animal species forming them, but from the special form of toxin, bacteria, etc., which were employed in treatment, and they are specifically attracted to the toxins, bacteria, etc., which are attacking the organism. Every organism possesses a certain degree of natural resistance at the moment when the disease commences, a resistance which, because of the attempts at protection, will usually increase during the disease, but which may decrease, whilst anti-substances are formed and brought into action. An infective disease, therefore, as a rule represents a very complicated process when regarded from the standpoint of immunity. The analysis of the individual factors of this combined process falls to the task of clinical medicine, and it is to be hoped that in the course of time not merely theoretical explanations, but also points of practical importance will be discovered.

[Recent Views on Immunity

Bactericidal and Hæmolytic Action.—It may be well again to reiterate the fact that in only a few of the diseases due to bacterial invasion is a soluble toxin formed. In most instances we have a rapid proliferation of the organisms within the body, and their action by the production of intracellular toxins. In the former group of infections cure may be brought about by a simple neutralisation of the toxin circulating in the blood, *e.g.* in diphtheria; in the second series what is essential is the destruction of the bacteria themselves.

The Pfeiffer Phenomenon.—Pfeiffer discovered that if an animal was immunised by inoculation with dead cholera vibrios, and if, thereafter, a lethal dose of living vibrios was introduced into the peritoneal cavity, the organisms were killed within a comparatively short period. He further showed that on injecting into a normal animal living cholera vibrios along with anti-cholera serum which had been heated to 58° C., and had in consequence no bactericidal power *in vitro*, the same destruction of the organisms took place. It was inferred from these observations that the anti-cholera serum did not in itself contain the bactericidal element, but that it merely, in some way or other, enabled the tissues of the non-immune animal to cope with the bacteria. The nature of the defensive mechanism was further elucidated by Bordet, who found that while no bactericidal action occurred in a mixture of heated anti-cholera serum and vibrios, the addition of fresh serum from a non-immune animal caused bacteriolysis, but that if the fresh serum had been previously heated it lost this power. Bordet thus proved that in the bactericidal action two substances are concerned: one, in the fresh serum, which is thermolabile, and one in the immune serum, which is thermostable. The thermolabile element we call the *complement* or *alexin*, the thermostable element is the *immune body*, *copula*, or *amboceptor*. The latter, moreover, is a specific substance—a mixture of fresh serum with inactivated anti-cholera serum being bactericidal to cholera vibrios only.

Hæmolytic Sera.—Bordet also pointed out that the phenomena following the inoculation of one species of animal with the red blood corpuscles of another species, present many analogies to the above, and it is on this account, as well as because blood corpuscles form a very convenient means of investigation, by reason of the ease with which hæmolysis can be detected *in vitro*, that so much work has been done on hæmolytic sera. If an animal A be immunised by repeated inoculation with the blood corpuscles of another species B, the serum of A acquires the power of lysing B's corpuscles both on intravascular injection and *in vitro*. The action is specific, or nearly so; that is, the corpuscles of another species are not affected. This immune, or hæmolytic serum, can, like a bactericidal serum, be inactivated by heating to 58° C., and can again be activated by the addition of some fresh serum from a normal animal, but not by adding fresh serum which has been heated. Hæmolytic sera, therefore, contain thermostable

immune body and thermolabile complement; fresh serum contains complement only.

The relations of immune body and complement were still further studied by Ehrlich and Morgenroth. The following experiments illustrate the methods employed in research of this nature:—A mixture of sheep's corpuscles and inactivated immune goat's serum was centrifuged, the fluid (A) decanted and the corpuscles (B) washed:—(1) A + sheep's corpuscles + fresh goat's serum = no hæmolysis; therefore the immune body present in the inactivated serum has been removed from it. (2) B + fresh goat's serum = hæmolysis; therefore the immune body of the inactivated goat's serum has transferred itself to the corpuscles. Having thus demonstrated that immune body interacts with corpuscles, the next step is to ascertain whether complement also will do so. A mixture of fresh goat's serum and corpuscles was centrifuged, the fluid (C) decanted and the corpuscles (D) washed. (3) C + sheep's corpuscles + inactivated immune goat's serum = hæmolysis; therefore the fresh serum still contained complement. (4) D + inactivated immune goat's serum = no hæmolysis; therefore the corpuscles have not attracted complement. These observations clearly demonstrate the dual nature of hæmolytic and bactericidal action. There are two partners in the work of bacteriolysis and hæmolysis—the specific immune body, copula, or amboceptor, present only in the serum of an animal which has been immunised, and the complement, or alexin, present in the serum of the normal, non-immune animal.

So far only artificially-produced immune bodies have been spoken of; it should, however, be stated that some occur naturally. Thus, for example, the serum of the goat hæmolyses the corpuscles of guinea-pigs and rabbits.

The question arises, Is there only one, or are there many, immune bodies concerned in such phenomena? Ehrlich has shown that the latter is the true state of affairs, and that it is possible by immunising animals with cells, both from different organs and from various species, to obtain a great variety of specific cytotoxic sera, each with its proper immune body. But when we come to ask the same question concerning the complement, we do not get so clear an answer. Ehrlich holds that complements, like immune bodies, are multiple; while Bordet, and the French school generally, adhere to the view that the complement is single. This and many other problems connected with hæmolytic and bactericidal sera as yet await solution, and the complexity of the questions at issue is such that it is impossible to give any concise summary of the opposing arguments or evidence which shall yet be comprehensible. All observers are agreed as to the actual facts described above, and at this we may leave the matter.

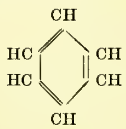
The essential facts of hæmolysis have been stated to hold good for bactericidal action so far as cholera vibrios are concerned; the same may be said of the organism of typhoid and dysentery, and we may justifiably imagine that the action of many other antibacterial sera, whether they are bactericidal or bacteriolytic, is also analogous. It would, however, be erroneous to suppose that all immunity rests on an increased bactericidal power of this nature. Wright's work on Opsonins (*vide* p. 373) shows that the organism is provided in addition with quite other lines of defence.

Ehrlich's Side-Chain Theory.—Although many criticisms have been levelled at it, the "side-chain" theory of Ehrlich may be said to receive general acceptance in its main outlines as a working hypothesis of the nature of immunity. It must be remembered that it is a theory only, and that it will probably require to undergo modification as new facts come to light. Many details are open to question, yet as it offers more than any other a fairly coherent explanation of many of the phenomena of immunity it deserves some consideration here.

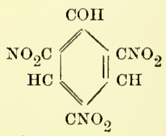
Ehrlich looks on the neutralisation of toxin by antitoxin as a chemical, not a physiological process, and in this the majority of observers agree with him. In standardising diphtheria-toxin against a standard serum of which a given quantity was able to neutralise 100 minimum lethal doses of toxin certain anomalies presented themselves. (1) A less quantity of toxin than 100 M.L.D. neutralised the serum, and (2) to a neutral mixture of toxin and antitoxin more than one extra M.L.D. of toxin had to be added in order to kill a guinea-pig within the conventional time. Ehrlich explained these anomalies by showing that in crude toxin some of the toxin molecules underwent modification to toxoids. These toxoids, though almost non-poisonous, could still combine with antitoxin. Their combining affinity might be equal to, greater than, or less than that of toxin. Now when a crude toxin containing toxin + toxoid of lower affinity is used to neutralise antitoxin, and subsequently more toxin is added to the mixture, the first molecules to be set free will be the toxoids, and until all these are liberated no free toxin will be present, and no poisonous effect will be produced. Thus variations in the number of M.L.D. of crude toxin required to neutralise completely a standard antitoxin must depend on the relative amount of toxin and toxoid present, while the fact that more than one additional M.L.D. is needed to convert the neutral into a lethal mixture arises from the greater affinity of toxin for antitoxin in preventing any free toxin being present so long as any combined toxoid exists in the mixture. The fundamental point to be grasped is the power of the molecule with stronger combining affinity to turn out that with the weaker affinity. From the above observations Ehrlich also assumed that the toxin (toxoid) molecule consisted of two groups, one

capable of uniting with antitoxin, the other poisonous (or but slightly so). The former he named the *haptophore*, the latter the *toxophore* group.

Ehrlich conceives the living cell as possessing a constitution analogous to the ring of the Benzene molecule, to which, as a centre, outlying molecules are attached. These outlying groups



Benzene.



Trinitrobenzene.

or molecules, side-chains or receptors, are supposed to play an important part in the nutrition and life of cell by combining with food molecules, oxygen, etc., circulating in the blood. They are, moreover, the mechanism through which toxins are able to act on the cell. The toxin molecule becomes attached by its haptophore group to the corresponding side-chain, whereupon the toxophore group comes into relation with the cell protoplasm. The cell may thereby be irretrievably damaged, or short of this, the side-chain alone may be destroyed. In the latter event the cell at once sets to work to produce new side-chains, and at last, as the result of repeated doses of poison, these are formed in excess of the cell's requirements, and are thrown off into the circulating blood. When the side-chain is free in the plasma and becomes attached to the haptophore group of a toxin molecule the toxophore group is rendered impotent by being deprived of the only means through which it can anchor itself to, and damage the tissues. Antitoxin, therefore, is simply blood serum containing large numbers of free side-chains.

The theory also admits of an explanation of the phenomena of hæmolysis, etc. When an animal receives a large enough dose of foreign red blood corpuscles, its metabolism is interfered with, and it may be killed. It is supposed that this takes place through the union of red blood corpuscles, or their products, with certain somatic cells. Should the dose be less, immunisation takes place, and, as has been shown, an immune serum containing complement and amboceptor can be obtained, the amboceptor, it will be remembered, possessing the power of attaching itself to red blood corpuscles. Like antitoxin, this immune serum owes its properties to containing cast-off side-chains which the attack of the red blood corpuscles has stimulated the body cells to produce in excess. These side-chains, therefore, constitute the immune body, and act by attaching themselves to red blood corpuscles on the one hand and complement on the other, whereby the latter is able to lyse the former. Immune body, therefore, acts as a copula and has two affinities—it possesses two haptophore groups, one *complementophile*, the other *cytophile*. The action of the complement is conceived of as resembling that of a ferment; it must also possess two groups—one concerned in the destruction of blood corpuscles, which is called the *zymophore*, the other, by which it anchors itself to copula, called again haptophore. Fischer suggests as a simile that if we consider the cell as a lock and the copula as a key, the complement is the hand which turns the key. Many hands may do this, but only one key will fit the lock (Fig. 1).

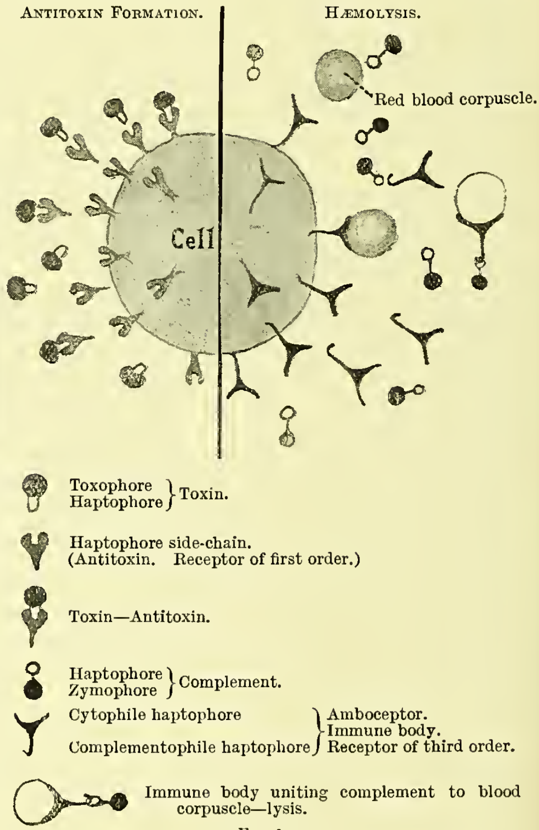


Fig. 1.

Biological Aspects of the Side-Chain Theory.—The chief implications of the theory, perhaps, concern its bearing on the nutritive processes going on in living protoplasm. Ehrlich considers that the mechanism in question is that through which food is assimilated by the cells, and when we remember how closely many of the bodies employed in producing immunity resemble foods the plausibility of the supposition is at once evident. According to the author of the theory, three kinds of receptors, or side-chains, play a part in the nutrition of the cell. First, there is the case of a food molecule ready for assimilation; a single haptophore group will anchor this. Such a *receptor of the first order* is analogous to the simple side-chains of which antitoxin is composed. If, however, the food molecule requires further preparation to render it

assimilable two possibilities are conceived of—(*a*) The side-chain may consist of a haptophore group and a group with digestive power, and this *receptor of the second order* may be compared with the agglutinins and precipitins which appear in the serum after immunisation. (*b*) The receptor may possess two haptophore groups, to one of which the food molecule anchors itself, while the other has an affinity for some molecule possessing digestive properties. These *receptors of the third order* find their analogy in the immune body which needs for its bactericidal or lytic action the coöperation of the complement.

Therapeutic Use of Bactericidal Sera.—The fact that for bactericidal sera to act, complement is essential, is fundamentally important. Let us suppose, for the sake of argument, that by inoculation we can greatly increase the immune body, and that we can inject this into a person invaded by the organism in question; we have still to face the fact that (beyond the small quantity which the immune serum contains) there is no more complement than formerly, and that the immune body is only available for the destruction of bacteria in so far as it can be complemented. Thus there is at once a practical difficulty, and it should be added that there is some reason to believe that an excess of immune body over available complement may defeat its own ends. For under these conditions a part of the immune body may attach itself to the bacteria and another part to complement, whereupon the only way in which complement and bacteria can come into relation is by immune body uniting to immune body, which is impossible. So far, it has not proved possible practically to increase the complement. For one thing, little is known as to whether complements are single or multiple, and the search for a suitable one is largely at random. Again, to inject complement may simply result in the formation of anticomplement, which will neutralise what already exists. Antibacterial sera originally contain the complement of the species from which they are derived, but complement is an unstable body and liable to disappear as time goes on. It does not follow that the complement present in man will be able to attach itself to the immune body injected; that, therefore, may prove useless. The whole question of complementing antibacterial sera may be said to be the crucial point on which their future usefulness seems to hinge.

Phagocytosis.—The most important recent advance in connection with the relations of what we may call cellular and humoral immunity is due to the researches of Sir A. E. Wright and his collaborators on Opsonins. They estimated the phagocytic power of the leucocytes by counting the organisms ingested by these cells when equal parts of a standard bacterial emulsion and human blood were incubated together at 37° for 15 minutes. They further devised methods by which the respective parts played by corpuscles and serum in the rôle of phagocytosis could be determined, and proved that "blood fluids modify bacteria in a manner which renders them a ready prey to phagocytes." To this influence they gave the name *opsonic action*, and to the constituents of the serum which so act, *opsonins* (ὀψωνέω, I prepare victuals for). The method adopted is to withdraw blood by a pipette, to centrifuge and decant off the supernatant serum, and to wash the corpuscles. The serum and corpuscles can then be tested separately against a standardised bacterial suspension. Wright, working with an emulsion of *staphylococcus pyogenes aureus* and his own corpuscles and serum, found that the number of organisms ingested fell when serum which had been kept for four or five days, or heated to 60°-65° for 10 minutes, was used. When the serum was first allowed to act on the bacteria, and then heated, and the mixture thereafter exposed to the action of the corpuscles, phagocytic action was unimpaired, whence it follows that serum contains some substance which so affects bacteria as to cause them to fall a ready prey to phagocytes. This opsonic power of the blood is exerted in regard to *staphylo-* and *streptococci*, *B. pestis*, *M. Melitensis*, *diplococcus pneumoniæ*, *B. coli*, Shiga's bacillus, *B. anthracis*, *B. typhosus*, *B. tuberculosis*, and the cholera vibrio. It is not exerted on *B. diphtheriæ* or *B. xerosis*. The nature of opsonins is uncertain. There is some reason to suppose that they are analogous to agglutinins and precipitins, and consist of a haptophore and an opsoniphore group.

By comparing the number of bacteria ingested in (*a*) a mixture of *normal corpuscles*, *bacterial suspension*, and *normal serum*, and (*b*) *normal corpuscles*, *bacterial suspension*, and the *serum of a patient suffering from a bacterial disease*, a ratio, called the Opsonic Index, is obtained. In cases of localised infection with staphlococcus aureus—*e.g.* sycosis, boils, etc.—Wright invariably found this ratio, or index, lower than normal—from ·1 to ·87, 1· being normal. The inoculation of such patients with a vaccine of dead staphylococci is followed, first, by a temporary fall in the opsonic index ("*negative phase*"), and then by a more prolonged rise ("*positive phase*") to or above normal. Coincidently with the rise in the opsonic index, there is a great amelioration of the lesion due to the bacterial invasion. In connection with the subject of bacterial invasion, Wright lays great stress on what he terms the *bacteriotropic pressure*—*i.e.* the mass effect exerted on the invading bacteria by the protective substances contained in the blood fluids. This does not stand at the same level throughout the organism. In fatal typhoid or Malta fever, for instance, the agglutinating action may be as much as 200 times less in the splenic pulp than

in the circulating blood. Bacteria live and multiply in these regions of lowered bacteriotropic pressure. Thus in the case of an abscess the opsonic power of the pus serum was found to be only one-sixth of that of the blood.

Wright's work on Opsonins has chiefly been made use of and extended in connection with tuberculous infection. Tubercle may be either localised, or general, and attended with constitutional disturbance. In localised tubercle the opsonic index is low; in the generalised form it may be either low or high, up to as much as twice normal. When the infection is active the opsonic index rises and falls within wide limits, because the patient is inoculating himself with successive doses of tubercle, and in consequence negative and positive phases are alternating with one another. The question naturally arises, Is the low opsonic index in localised tuberculous and staphylococcal infections a cause, or a result, of the bacterial invasion? Wright is of opinion that it is an evidence of failure of the defences of the organism, and that to it the infection is due.

The diagnostic significance of the opsonic index in tuberculosis may be stated thus:—(1) Persistently low opsonic power, with evidence of a local bacterial invasion, is a sign of tubercle. (2) Normal opsonic power with a local bacterial invasion excludes tubercle. (3) Fluctuating opsonic power indicates active tubercle. (4) If heating a serum of which the opsonic power is 1· does not inactivate it to the same degree as normal, it indicates either active tuberculosis or vaccination with tuberculin. (5) If the opsonic power of the fluids from the focus of invasion differs from that of the blood, tubercle is indicated; if it is the same, the infection is non-tuberculous.

Technique of the Estimation of the Opsonic Index.—Blood is collected in a pipette, shaped as in the diagram (Fig. 2); the ends are sealed and the tube is hooked by its curved end to the centrifuge. On centrifuging the corpuscles collect at *a*, the tube is broken across at *b*, and the serum pipetted off. Serum free corpuscles are next obtained by allowing some of the observer's blood to drop into a solution of 1 per cent sodium citrate in normal saline. The mixture is thoroughly centrifuged and the supernatant fluid decanted off; more saline is added, the mixture is again centrifuged, and the process is repeated once more. After this second washing the upper layer of corpuscles, which is rich in leucocytes, is employed as "washed corpuscles." Next an emulsion of the organism to be used is made by pouring some one or two per cent saline solution on an agar slope on which there is a culture. This is allowed to settle, the upper part is centrifuged to remove clumps of bacteria, leaving a slightly opalescent supernatant fluid—the bacterial suspension. The tubercle emulsion is made by grinding up a loopful of dead bacilli in an agate mortar with salt solution, and then centrifuging. Having made these preparations, we mix equal parts of serum, blood corpuscles, and emulsion, using as a measure a capillary tube with a mark about an inch from one end, and provided at the other end with a rubber teat. The serum is drawn up to the mark, and then an air-bell is allowed to enter, next the emulsion is sucked up, and another air-bell, and then the corpuscles. The contents of the tube are then blown out on to a slide, thoroughly mixed, and re-aspirated into the tube once more. After being sealed, the tube is incubated at 37° for 15 minutes. A control preparation containing normal instead of pathological serum is made and treated in the same way. From the two tubes films are made and stained in the usual manner. The number of bacteria in 20 or 30 cells is counted, and an average struck. The ratio of the number ingested in the preparation with the patient's serum to that in the control, stated as unity, gives the opsonic index, *e.g.*—

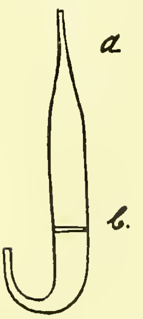


FIG. 2.

Patient's serum + washed corpuscles + bacterial suspension. Average bacteria 1·5.

Normal serum + washed corpuscle + bacterial suspension. Average bacteria 3·. Opsonic index $\frac{1·5}{3·}$ or ·5.

Antituberculous Inoculation.—Wright advises the use of very small doses of Tuberculin R.—$\frac{1}{1000}$ to $\frac{1}{600}$ milligram. The remedy should only be employed when there is evidence that the infection is localised, as shown by a persistently low opsonic index. Following upon inoculation there is the usual negative phase, succeeded by the positive phase. It is important not to repeat the dose during the continuance of the former. Practically the tuberculin should not be given at intervals of less than 10 days. Wright finds that it is not possible to produce a cumulation of positive phases by re-inoculating during the positive phase. On the other hand, it is easy to produce a cumulation of negative phases by re-inoculating during the negative phase. Hence he prefers to regard each inoculation as a separate event, and to be satisfied if ultimately the opsonic index can be raised to the normal level.

Antistaphylococcus Inoculation.—A considerable measure of success has attended the treatment of localised staphylococcal injections with a vaccine consisting of a sterile culture of staphylococci. The amount inoculated is from ·5 to 1 cc. increasing to 2 cc., and the vaccine is standardised so that each cc. contains 2,500,000,000 staphylococci. Injection is followed by a negative, and then a positive phase. It is possible by using this vaccine to raise the

opsonic index considerably, and to maintain the high level for a long period. The injections are repeated at intervals of a week or a fortnight.]

LITERATURE.—A record of current literature is contained in:—BAUMGARTEN. *Jahresbericht d. path. Mikroorganismen.*—KOCH and FLÜGGE. *Zeitschr. f. Hyg. u. Infects.*—*Centralb. f. Bakt. u. Parasit.*, Abthl. I.—MALY. *Jahresbericht ü. d. Fortsch. d. Thier-Chemie.*

Impaction.—The condition of being firmly fixed or impacted, as when fæces in large amount become fixed in a portion of the bowel (*fœcal impaction*).

Impaludism.—The morbid condition of body in which there is a special tendency to become affected with malaria (Lat. *palus*, a marsh). *See* MALARIA.

Impar.—Unequal, odd (as opposed to even); used in the description of structures (*e.g.* some arteries and nerves) which are not paired; an azygous part.

Imperforation.—The condition of being imperforate, *i.e.* of showing no opening when normally an opening should be present; the closure of the opening may be congenital or acquired; atresia. *See* MENSTRUATION AND ITS DISORDERS (*Retention of the Menses, Atresia of Hymen or Vagina*); UTERUS, MALFORMATIONS OF (*Atresia of the Genital Canal*).

Imperial Drink. *See* INVALID FEEDING (*Food in Pyrexial States*).

Impetiginodes. *See* IMPETIGO.

Impetigo. *See also* ALOPECIA (*Varieties*); DERMATITIS HERPETIFORMIS (*Diagnosis*); PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Cutaneous Rashes, Impetigo Herpetiformis*); SKIN, BACTERIOLOGY OF (*Impetigo*); SYCOSIS (*Impetigo Contagiosa, Impetiginous Eczema*).—The term impetigo (*ab impetu*) is applied to an eruption, or group of eruptions, characterised by the evolution of small superficial epidermic vesicles, vesico-pustules, or pustules of the kind formerly described as psydracious, and distinguished clinically from the larger ecthymatous pustule, the acneiform perifollicular pustule with indurated base, and from the sycosiform pustule. Not all pustular eruptions are, however, impetigo. There is a rare, primary, pustular disease of a totally different nature known as *impetigo herpetiformis.* In all the vesicular and bullous eruptions the contents of the lesions may become puriform, *e.g.* vesicular eczema, dermatitis herpetiformis, herpes. On the other hand, a host of pruritic eruptions may be complicated by pus formation following scratching, *e.g.* eczema, animal parasitic diseases, prurigo, lichen urticatus, prickly heat. As the pus formation thus secondarily complicating eruptions is probably due to microbes similar to those producing the impetigo under consideration, it is evident that the subject is a very complex one. It is at the present moment being subjected to rigorous investigation at the hands of bacteriologists.

The dismemberment of the Willanean groups *porrigo* and *impetigo*, and the transference of the constituents to the pustular or impetiginous phases of eczema, left one variety to be dealt with, characterised by contagious and auto-inoculable "favous pustules" (*porrigo favosa* of Willan and A. T. Thomson, *impetigo favosa* of Bateman), and even this was wrongly included under eczema by Hebra, and confounded by Bayer and Biett with favus on account of the name. Startin, senior, whose name is frequently mentioned in connection with the history of the eruption, identified this affection with one he had christened *porrigo simplex* (*Medical Times*, 1846), and Devergie in France also recognised its contagious nature. In 1864 and 1869 Tilbury Fox attracted general attention by his papers on *impetigo* or *porrigo contagiosa*, and thenceforward the literature multiplied. Fox described a *vesicular* eruption, primarily of systemic origin and tending to run a definite course, but kept up by secondary auto-inoculation. He proved the inoculability experimentally, and was corroborated by van Harlingen. This definite picture was somewhat blurred by the further observation that similar phlyctenæ might arise out of and around vaccination crusts, about cuts and like injuries, and that it might complicate eczema, scabies, and other affections, and *vice versa*. He expressly states that pediculi were absent as a rule, and in his *Atlas* (pl. xxiii. xxiv.) he differentiates between a pustular eruption, which is non-contagious and often associated with pediculi in the head, and his vesico-pustular impetigo contagiosa. The former frequently complicates the latter, he said, and so gave rise to confusion. On the other hand, J. Hutchinson, who was familiar with Startin's teaching, figured (*Syd. Soc. Atlas of Skin Diseases*, pl. xv. xx.) *common contagiosa porrigo* almost universally, but "probably accidentally," associated with pediculi capitis, and characterised by the formation of vesications, which dry into thick, dirty pus scabs. Extant drawings of a similar eruption secondary to vaccination crusts are alluded to. He thought the eruption produceable on an irritable skin by any kind of local irritation, and thence inoculable on other parts and on other persons by scratching. Coming to later times, Radcliffe Crocker holds that no line can be drawn between the epidemic febrile form of Tilbury Fox and the common non-febrile, more localised, cases of less definite course, which may have their origin in the effects of scratching, and are always due to the inoculation of contagious *pus* independently of its source. F. J. Payne characterises the lesions as vesico-pustules, and states that the original source of infection is always some kind of *suppuration*, such as the pus formation in pediculosis capitis, old vaccination scabs, conjunctivitis, otorrhœa,

purulent rhinitis, ulcerative stomatitis, vaginitis, festered wounds or scratches; and many of these in turn may be secondary to impetigo.

We thus see that *impetigo* is the name given by most authors to an eruption of superficial vesicles, vesico-pustules, or pustules, arising from the inoculation of corresponding contagious exudations, and that for the most part the vesicular eruption described by Tilbury Fox is not distinguished as a separate affection from the primarily pustular phases, although his name *impetigo* (*porrigo*) *contagiosa* is widely adapted to include all. Duhring, however, distinguishes a primarily pustular impetigo. It must be clearly understood that in addition to the primary cases originating in the sources detailed by Payne, others in succession may be derived by direct or indirect inoculation,[1] and appear to be spontaneous, *i.e.* their source cannot be identified. Further, that similar eruptions, generally pustular, but differing as a rule in their wider dissemination, may appear as complications of a host of pruritic eruptions.

The all-important *bacteriological aspect* has next to be considered. It has been established that for the most part the staphylococci are the causal agents of pus. Other organisms, however, are also pyogenic, such as the bacilli of typhoid fever, glanders, tuberculosis, soft chancre, actinomycosis, and some ringworm fungi of animal origin. In 1887 an important paper by Bockhart appeared (*Monatsh. f. prakt. Derm.*), in which he proved that a form of impetigo with primary pustules, generally found about the nates and extremities, and often secondary to pruritic eruptions, was caused by staphylococci. He regarded furuncle and sycosis as different forms of the same disease. Since then it has been generally taught that impetigo, in the wide sense, is a staphylococcia. Soon observers were forthcoming who affirmed the streptococcic origin of some forms, and now the battle rages over this difficult problem. The latest author, Sabouraud, contributes (*Ann. de Derm. et de Syph.*, 1900) an elaborate memoir, in which he insists on the utter confusion which has arisen from not recognising a distinction between the streptococcic vesicular or phlyctenular impetigo contagiosa of Tilbury Fox and the staphylococcic pustular peripilar impetigo of Bockhart. This may be so, but putting on one side the latter eruption, many of us are still puzzled to know if we rightly include with the apparently spontaneous vesicular forms the more or less pustular phases arising from the various local sources detailed above. We may here note that the organisms in dispute are constantly present in the skin, and Payne supposes that "they in the first place cause inflammation, if the tissues in which they live are injured in any way; then growing as a pure cultivation in the inflamed tissue they acquire increased pathogenetic properties, and, by passing through one focus of inflammation after another, ultimately acquire sufficient virulence to cause suppuration in healthy skin." He notes that pus from deep abscesses does not appear to produce impetigo, and suppurative acne rarely.

Impetigo contagiosa of Tilbury Fox (vesicular or phlyctenular impetigo) is a very common eruption, occurring especially in children and in the poorer classes, but occasionally seen in adults. It is auto- and hetero-inoculable directly and indirectly, sometimes quasi-epidemic, and varies from time to time in frequency and virulence. Sabouraud affirms that this eruption is due to a streptococcus, and the same which causes erysipelas, though Unna and Schwenter think it distinct. He points out that, if an ordinary blister be raised on the skin, hardly has the vesication risen before it is invaded by microorganisms, and so it is with the impetigo vesicle. The streptococci are scanty and difficult to cultivate in the early stages, and are rapidly mixed with a secondary staphylococcic infection.[2] Now the pure streptococci impetigo is characterised by the evolution of tiny, *isolated*, inflammatory papules, which rapidly vesicate, or of clear vesicles, of a uniform type, without relation to a hair follicle and unaccompanied by any marked local disturbance of sensation. The vesicle remains clear as long as the causal streptococci culture continues pure, but becomes opalescent and pustular by leucocytic invasion consecutive to secondary staphylococcic infection. Secondary inoculations may contain mixed organisms from the first and furnish primary vesico-pustules, and, if the secondary staphylococci invasion be strong, even true pustules. The vesicles or vesico-pustules tend to enlarge into flat phlyctenæ, attaining in five or six days the size of a sixpence or shilling, unless ruptured by scratching, which generally occurs promptly. Even then a characteristic extending vesication can be often seen. The phlyctenæ are rarely full and pemphigoid (see Tilbury Fox's *Atlas*), sometimes simulate a vaccine vesicle, but generally are flat and collapsed. As in all excentrically spreading eruptions ringed or marginate lesions are not infrequently formed, and in rare case the prevalence of this type and their confluence produce strikingly figured pictures. The contents of the phlyctenæ dry up into crusts, often with a stuck-on appearance. If the contents are serous the crusts will be amber-coloured, but if puriform more or less thick and deeply coloured. The isolated lesions are often scanty, sometimes copious, and the crusts may be crowded in parts so as to form confluent patches simulating those of pustular eczema. The fall of the crust leaves a temporary red macule, which disappears without scarring. The initial evolution is generally

[1] *E.g.* by towels, pillows, washing flannels, antimacassars, football jerseys, etc.

[2] We must refer the reader to Sabouraud's memoir for a description of his special methods of cultivation.

local and on some exposed part, especially the face, or in the neighbourhood of the original source of the contagious exudation. Thence it may be inoculated on any other part, particularly the fingers, and in rare cases the greater part of the surface may be involved, and pemphigus be simulated. The mucous membrane of eye and nostrils may be implicated. On the limbs the lesions tend to assume the more formidable aspect known as *ecthyma*, and on the hands the vesications may attain a large size owing to the thicker corneous layer. I have seen a bag of serum hanging from a finger. Perionychia is not uncommon. True staphylococcic inoculations, such as we are about to describe, may also occur as complications, *e.g.* suppurative folliculites and even furuncles. The related glands may swell, but rarely suppurate. Tilbury Fox and others described an invasive febrile period, and a natural short and definite course, generally masked by the auto-inoculation of fresh lesions. The affection, however, is to be regarded as primarily a local one. I have twice seen a scarlatiniform rash arise from secondary intoxication.

It is probable that the so-called *pemphigus neonatorum*, sometimes epidemic in lying-in institutions, and a *contagious* "*pemphigus*" observed by Manson and others in tropical regions, belong here. Such infections own obvious sources, or may be apparently spontaneous, and they complicate a host of pruritic eruptions, and must be carefully distinguished from the original trouble. The ordinary localised phases of impetigo contagiosa are distinguished from eczema by the successive evolution of isolated lesions, without marked sensory disturbance, their tendency to peripheral extension and the formation of amber crusts of the discharge remain serous. Confluent areas present a real difficulty, but the presence of typical isolated lesions around generally affords a clue.

The IMPETIGO OF BOCKHART is described by Sabouraud as of frequent occurrence and as an entirely distinct morbid entity due to the staphylococcus aureus, which, unlike the streptococcus, remains uncontaminated by other organisms. The typical lesion is a superficial pustule *ab initio* of greenish-yellow colour, centred by a hair, and surrounded, especially the younger ones, by a red areola. Such pustules may be of all sizes up to a pea, or even the end of the little finger, and they may be disseminated or crowded over the whole or part of a region, especially hairy regions, such as the scalp of the child, the moustache and beard of man. They may, however, occur anywhere. The pustules terminate in crusts, and when the latter fall the orifice of the follicle appears red and inflamed, and a deeper-seated folliculitis or a furuncle may result. Inoculation about the nail may set up paronychia and onychia. Striking characters are the frequent returns of the eruption, its sudden evolution in some hours, and the premonitory pain in the related lymphatic glands. This eruption can arise without obvious cause, or be secondary to all sorts of traumatisms, such as medicinal plasters, or a wet dressing, or such as happen to washerwomen, masons, and printers from various chemical substances. It can also complicate pre-existing affections, such as acne, the impetigo of Tilbury Fox, eczema (E. impetiginodes), prurigo, lupus, etc. This impetigo is auto- and hetero-inoculable, but there does not seem to be the tendency to excentric spread seen in the streptococcic impetigo.

Diagnosis. — The fundamental distinction between the staphylococcic pustule developed about a hair follicle, and the streptococcic phlyctena, without special relation to the hair follicle, which becomes complicated by staphylococci, and hence leads to mixed infections, has been sufficiently pointed out.

Treatment.—The recommendation of a multitude of remedies usually signifies difficulty of cure, but not in the present instance. The principles are to cleanse away, and, as long as new formations arise, to continue to remove, all infective vesicles or pustules, discharges or crusts. The latter should be softened by boric acid fomentations, carbolised vaseline, carbolic oil, and so on, and then thoroughly bathed away as fast as they form. All vesicles and pustules should be ruptured, and the infective exudations disinfected and removed by antiseptic washes made with boric or carbolic acid, or the biniodide or bichloride of mercury, and such like. The raw, exuding areas may be dried up by parasiticide and astringent lotions, or by mulls or plasters, or better still by ointments or pastes of oxide of zinc, to which 2 or 3 per cent of some parasiticide or antiseptic has been added, such as ammoniated mercury, naphthol, sulphur, carbolic acid, or salicylic acid. The following formulæ will serve as illustrations:—Sulphate of zinc 7 grammes, sulphate of copper 2 grammes, distilled water saturated with camphor and filtered 600 grammes. Make a lotion. Use to arrest early lesions (Sabouraud). Plumbi acetatis 1·0, acidi salicylici 2·0, zinci oxidi 20·0, adipis 50·0, petrolate 50·0. Infr. ung. (Dubreuilh). Acidi carbolici gr. v., acidi salicylici gr. x., pasta zinci oxidi ℥j. M. ft. pasta (Duhring). Hydrarg. ammon. gr. xij., ung. zinci benz. ad ℥j. M. ft. ung. (Tilbury Fox). Original sources of infection, such as purulent inflammation from mucous membranes, pediculi capitis, suppurating vaccination lesions, etc., must be sought for, and if they exist, cured. Lastly, not only must direct sources of inoculation be provided against, such as scratching, but indirect, such as soiled towels, pillows, chair and sofa coverings, caps, football jerseys, razors, and so on.

In the rarer, more or less universal cases, especially those sometimes seen in babies, the prognosis may be grave, unless the skin is thoroughly disinfected by prolonged antiseptic

baths. The case has to be treated like a bad burn.

Impetigo Herpetiforme

Under the titles *Impetigo herpetiforme* (Hebra), *Dermatite pustuleuse circinée et excentrique* (Besnier and Doyon), *Infection purulente tégumentaire* (Hallopeau), a rare malady of much gravity has been described, characterised by the formation of superficial pustules, tending to arise in patches spreading excentrically, which invades a great portion of the skin and evolves incessantly by successive outbursts accompanied by fever. At first it was supposed to occur exclusively in gravid females, like *herpes gestationis*, but in later years it has been described in women apart from pregnancy and also in man. The eruption has many analogies with the pustular phases of dermatitis herpetiformis, but in the latter the pustules are mostly formed, often very rapidly, out of vesicles; in I. herpetiforme the pustules evolve as such. Whether the eruption is due to a pyæmia, or toxæmia, or a neurosis, is not certain. Kaposi has published some striking portraits (*Archiv für Dermat.* 1887).

Implantation.—The process of grafting or inserting, or the thing so ingrafted or inserted. Implantation cysts are such as form round foreign matter accidentally introduced into some part of the body (*e.g.* the eye). The name is also given to that form of monstrosity in which one fœtus is found in the interior of another (*fœtus in fœtu*).

Impotence. *See* Scrotum and Testicle, Diseases of (*Impotence*); Morphinomania (*Effects*); Obesity (*Etiology*, *Castration*); Sterility.

Impregnation. *See* Fœtus and Ovum, Development of (*Fertilisation*); Sterility.

Impressions, Maternal. *See* Maternal Impressions.

Impulse.—A thrust or beat (*see* Physiology, Circulation, *Cardiac Impulse*); also a "sudden access of volition" (*see* Insanity, Nature and Symptoms, *Mental Functions, Impulse*).

Impulsive Tic.—A chorea-like affection occurring in children, and consisting of involuntary muscular movements, explosive utterances (*echolalia*, *coprolalia*), and sometimes of fixed ideas; Giles de la Tourette's Disease.

Inadequacy.—Insufficiency, *e.g.* when the kidney or the thyroid gland is not active enough to perform its proper functions (renal or thyroid inadequacy).

Inanition.—The condition of exhaustion arising from want of nourishment, either from starvation or from inability of the organs to assimilate.

Incarceration.—The retention of an organ of the body in a place where it either ought not to be or from which it ought to have passed at an earlier time, *e.g.* the bowel in a hernial sac, the pregnant uterus in the pelvis, etc.

Incised Wounds. *See* Medicine, Forensic (*Varieties of Wounds*).

Incisor Teeth. *See* Children, Development of (*Dentition*); Teeth.

Inclusion.—The shutting up of one structure or body within another, *e.g.* fœtus in fœtu, or fœtal implantation, in which one fœtus is contained within another (in its abdominal or cranial cavity).

Incoherence. *See* Insanity, Nature and Symptoms.

Incompatibility. *See* Prescribing.

Incompetence.—A defect in the performance of the functions of any part of the body, but especially of the valves of the heart when they close imperfectly and allow the generation of a backward current in the circulation. *See* Heart, Affections of Myocardium and Endocardium; Pulse; etc.

Incontinence of Fæces.—Inability to control the expulsion of fæces from the lower bowel, as in torn perineum, etc.

Incontinence of Urine.—Inability to retain urine in the bladder. *See* Micturition (*Incontinence*); Prostate Gland (*Hypertrophy*, *Symptoms*); Urination, Disorders of (*Incontinence*).

Incoördination.—The results of want of harmony between the will power and the action of the muscular system. *See* Paralysis (*Introduction*); Spinal Cord, Medical (*General Symptomatology*); Tabes Dorsalis (*Symptomatology*, *Disturbance of Gait, etc.*).

Incubation Period. *See* Diphtheria (*Clinical History*); Disinfection (*Periods of Incubation and Infectiveness*); Epidemiology; Measles (*Incubation*); Scarlet Fever (*Stages*); etc.

Incubator.—An apparatus in which eggs, germs, or the new-born infant can be placed and kept at a constant temperature; a couveuse (*q.v.*). *See* Labour, Operations (*Induction of Premature Labour*); Sclerema Neonatorum (*Treatment*).

Incudectomy.—Excision of the incus from the middle ear.

Incus. *See* Physiology, Senses (*Hearing, Middle Ear*).

Indecent Assault. *See* Medicine, Forensic (*Rape*).

Index. *See* Anthropometry; Ethnology.

India. *See* Balneology (*India*).

Indian Drugs.—In the Indian and Colonial Addendum to the British Pharmacopœia of 1898 a good many drugs growing in and used in India are described, *e.g.* Indian Chiretta (*Andrographis*), Indian Orange Peel (*Aurantii Cortex Indicus*), Indian Gamboge (*Cambogia Indica*), Indian Gum (*Gummi Indicum*), Indian Oil of Verbena (*Oleum Graminis Citrati*), Indian Podophyllum, Indian Squill (*Urginea*), and Indian Valerian.

Indiarubber. *See* Caoutchouc; Trades, Dangerous (*Bisulphide of Carbon Poisoning*).

Indican.—Indoxyl-sulphate of potassium ($C_8H_6NSO_4K$), derived from indoxyl (C_8H_7NO), which in its turn is got by oxidation from indol (C_8H_7N). *See* Physiology, Excretion (*Urine, Sulphur-containing Bodies*); Urine, Pathological Changes in (*Colour*); Adrenal Bodies, Addison's Disease (*Symptoms, Urine*).

Indicanuria. *See* Urine, Pathological Changes in (*Aromatic Substances, Indicanuria*); Stomach and Duodenum, Diseases of (*Ulcer of Stomach, Special Symptomatology, Urine*).

Indies, West. *See* Therapeutics, Health Resorts (*Climates for the Aged, West Indies*).

Indigestion.

See also Insanity, Nature and Symptoms (*Etiological Varieties*); Nose, Accessory Sinuses, Inflammation of (*Indigestion from swallowing Secretions*); Rickets (*Etiology*); Stomach and Duodenum, Diseases of (*General Etiology, Infective Diseases*); Stomach and Duodenum, Diseases of (*General Symptomatology, Indigestion*).

The term indigestion serves as a cloak to cover a multitude of dyspeptic disorders, some of which come throughout their entire course under the name, others only so long as clear evidence of the presence of a distinct morbid lesion is wanting, constituting the primary cause, and worthy of consideration in the light of an individual entity. Indigestion, in truth, applies simply to symptoms of digestive unrest of objective or subjective character, denoting presence of a morbid condition of the tissues and cells in some part of the alimentary system itself, or in other systems of the body, and reflexly influencing digestive organs and processes through the mediation of the nervous system or the circulation.

Thus patients suffering from symptoms common to early stages of gastric ulcer, cancer of the stomach, or dilatation of that organ, from the results that neurasthenia often occasions upon the processes of peptic digestion, those of a gouty habit may give rise to, or that may spring from changes in the composition of the blood and alterations in its supply, are as truly the victims of indigestion as those affected by simple gastric catarrh. Unlike the majority of diseased types indigestion cannot, therefore, be associated with any one principal causal agent. Nor can the individual symptoms even be considered as indicative of one or other separate pathological condition. Functional faults and organic error are so closely and intricately connected in result, and so surely exercise some influence on each other, that where the one is the other generally exists also.

Classification

I. Acute.
- A. From Organic Lesions.
  1. Acute Gastritis.
  2. Gastric Ulcer (occasionally).
  3. Gastric Cancer ,,
- B. Through Nervous Influences.
  1. Cerebral.
  2. Spinal Gastric Crises.
  3. Peripheral.
- C. Toxæmic.
  1. Autogenetic.
     - (*a*) Uræmia, Cholæmia.
     - (*b*) Auto-toxæmia.
  2. Heterogenetic.
     - (*a*) Alcohol.
     - (*b*) Chemical Drugs.

II. Subacute and Chronic.
- A. From Organic Lesions.
  1. Simple or Chronic Gastritis.
  2. Gastric Ulcer (occasionally).
  3. Gastric Cancer.
  4. Dilatation of Stomach from Pyloric Stenosis.
  5. Gastric Cirrhosis.
  6. Tubercle.
  7. Syphilis.
  8. Venous Congestion.
- B. Functional.
  - (I.) Of Local Origin.
    1. Secretory.
       - (*a*) Acid.
         - (1) Hyperchlorhydria.
         - (2) Gastroxynsis.
         - (3) Hypochlorhydria.
       - (*b*) Ferment.
         - (1) Hypopepsia.
         - (2) Apepsia.

2. Sensory.
    (a) Sensation.
        (1) Heartburn.
        (2) Pain after or before food; Gastralgia.
    (b) Sensibility.
        (1) Tenderness on pressure.
        (2) To peristaltic movements.
3. Motor.
    (a) Hyperperistalsis.
    (b) Hypoperistalsis.
    (c) Pyloric Insufficiency.
    (d) Pyloric Spasm.
    (e) Dilated Stomach.
    (f) Gastroptosis.
    (g) Delayed emptying.

(II.) Of Distant Origin.
1. Cerebral.
    (a) Hysteria, Neurasthenia.
    (b) Hemicrania.
2. Spinal.
    (a) Spinal Irritation.
    (b) Some cases of Systemic Disease.
3. Peripheral.
    (a) Intestinal Lesions.
    (b) Pelvic Disease.
    (c) Pregnancy.
    (d) Enteroptosis.

C. Toxæmia.
1. Autogenetic.
    (a) Uræmia, Cholæmia, etc.
    (b) Auto-intoxication.
    (c) Gout and Rheumatism.
2. Heterogenetic.
    (a) Alcohol.
    (b) Drugs and Poisons.

The classification of cases of indigestion introduced above must not be regarded as being in any way either exhaustive or complete. Space forbids a further elaboration. As it stands it is meant to indicate the mode of origin and the nature of the primary cause of the various chief types of indigestion, apart from the many forms in which more than one of these factors coexist, and from those in which different symptoms are produced by similar causes. Every case of indigestion of organic source must necessarily exhibit some disturbance of function.

Those disorders of digestion which are purely functional from the first may continue without establishment of any organic change for some length of time, but if very prolonged generally induce actual changes in the tissues and cells of the stomach walls.

It cannot be too strongly insisted on that indigestion must on every occasion be regarded merely as a symptom, not as a disease to be treated by rule of thumb with routine remedies and stereotyped therapeutic measures. Few cases are identical in form, origin, or personal idiosyncrasy. In many the primary source from which the symptoms proceed has no direct relationship to the stomach or its digestive processes,—the morbid symptoms, subjective or objective, exhibited by that organ depending entirely upon irritation of the nervous tissues supplied to more or less distant parts, and consequent abnormal stimulation reflexly conveyed to the gastric nervous mechanism.

Again, constitutional affections may be really answerable for the occurrence of the symptoms, and unless they are dealt with along with the treatment of the local complaint, may inhibit success. In anæmia, gout, chronic debilitating diseases, little benefit will accrue unless the larger lesion is recognised as the more important factor in the management of the less.

Another class of case confesses to toxæmia as the *causa causans*; autogenetic when produced by excessive absorption of poisonous products of intestinal fermentation, proteolysis, or putrefaction, or by inability on the part of the liver to render the quantities conveyed to it innocuous; when aroused by presence of the results of metabolism in the blood-stream, because of renal inefficiency, or of biliary items where their passage into the bowel is arrested; heterogenetic when induced by alcoholic excess, or by ingestion or introduction of various gastric irritants into the stomach or body.

The general neuroses also must be borne in mind,—hysteria, neurasthenia, and mental depression,—in the consideration of indigestion; and in women, the wonderful influence exercised by diseased conditions of the organs of generation upon digestive processes, especially of the ovaries, as well as the intimate connection which the menstrual functions and pregnancy bear to them.

Still another point worthy of attention relates to the tendency that exists in cases in which certain glandular secretions, for whose real significance in the bodily economy we are still groping, have been arrested, or altered,—as, for instance, the secretion of the thyroid gland in myxœdema and goitre, or of the ovaries after double ovariotomy,—to the occurrence of dyspeptic troubles. Recognition of the fault underlying such cases will elucidate many of their puzzling symptoms, and indicate the most advantageous line of treatment.

Further, as "digestion" signifies all the processes by means of which the nutritive elements of the food introduced into the body are adapted for absorption into the circulating fluids for use, for assimilation, and in metabolism, so "indigestion" may be held to include many more recondite morbidities connected with the retail department of co-operative protoplasmic commerce, than the wholesale division, dealing with crude material, can offer. From the time

food is taken, up to the evacuation of that which is waste, and until the nutritious materials separated from it have been finally transformed into cell components, or paid their way in the production of energy at the cost of their own decomposition, digestion continues and indigestion is possible.

Robin's classification divides dyspepsias into three groups: dyspepsias from exaggeration of function, or hypersthenic; those due to insufficient function, or hyposthenic; and those caused by perversion of function, or gastric fermentations. Einhorn recognises organic disease with constant lesions, functional disease with variable lesions, and nervous affections of the stomach.

Etiology.—Indigestion respects neither age nor sex; male and female, old and young are liable to be attacked. Both age and sex, however, have some influence over the incidence of the malady, and the type.

Predisposing Causes. — 1. *Dietetic.* — Undoubtedly the chief source of indigestion lies in errors of diet. It is a very difficult and hazardous matter to define what constitutes a dietetic error in the universal sense. One man's meat is another's poison. Use and wont within certain limits influences the question. Habits, occupation, temperament, constitution, and bodily condition modify the conception of what may be appropriate in each separate case. Idiosyncrasy also has to be remembered.

Mere personal errors, apart from these considerations, consist in habitual omission to properly masticate the food, in hurried bolting of meals, immoderate use of alcoholic stimulants, of astringent infusions, ice-cold liquids, or over-hot drinks. In addition, where the circumstances of the patient place no restriction in the way of his or her indulgence of appetite, the continual enjoyment of highly spiced foods, the ingestion of one of the three primary forms of food-stuffs out of all proportion to the other two, whether it be proteid, carbohydrate, or fat, and a too close sequence of heavy meals, tend in time to produce dyspeptic symptoms, either from local quarrels on the part of the digestive organs, surfeited by excess, or from general conditions, reflexly causing a disordered state of the alimentary tract.

Indigestion from long-continued dietetic error, whether it be due to personal misdemeanours, to constitutional states, or to environment, chiefly arises from incongruity between the food taken and the physical needs of the body;—the amount of active expenditure of muscular energy, by inclination or of necessity, corresponding to but part of the nutriment ingested and absorbed;—or from unsuitable characters of the food for the upkeep of function—mental, secretory, or trophic. In those to whom circumstances deny a choice, irregular, unsuitable, and insufficient forms of sustenance, badly prepared or not prepared at all, often initiate chronic indigestion.

2. *Habits.*—Any ill effects which may follow such dietetic errors as are enumerated above are dependent in great part on the habits of the individual. Indiscretions in diet, productive of serious disturbance in the digestive economy of the sedentary occupant of an office, or of one largely confined by necessity to the house, may prove harmless to those who live an outdoor life, or whose occupation entails a considerable output of physical energy. But conversely, a monotonous or barely sufficient diet may serve for one who expends but little muscular force, while quite inadequate for the worker and a source in him of digestive disorder. A very common starting-point for indigestion is shown by persons, accustomed previously to an out-door life or much open-air exercise, who take up their abode in towns, or enter upon confined or sedentary occupations. The dietetic habits formerly suited to their circumstances are too often continued under these changed conditions, and if long maintained almost certainly result in ill-health.

Symptoms.—Owing to the fact that many of the clinical signs and symptoms of indigestion are common to more than one of its different types, perhaps the most simple and intelligible course to follow is to discuss them *seriatim*, as they fall under (1) Sensory, (2) Motor, and (3) Functional groups.

1. *Sensory Symptoms common in Indigestion.*—Local sensations of discomfort in the stomach are generally the first signs given of the presence of gastric disorder. They may only amount to a vague feeling of uneasiness, of a ball in, or some apparent weight pressing down, the stomach; again, they may take the form of a gnawing pain, persisting for some time, and usually directly related to the ingestion of food, or of more paroxysmal attacks of pain, either at some special stage of gastric digestion, or independent of digestive processes. Not infrequently the subjective sensation of pain is more or less definitely confined to one point, and can be located by the patient. In the majority of cases, however, the feeling of uneasiness and pain is diffuse and referred to the entire organ. Another common complaint is that of "swelling" of the stomach, a feeling as if it were greatly distended, and had too little accommodation for its bulk. This may arise from actual distension of the stomach with gas, or occur apart from any real dilatation or obstruction, and due to a hyperæsthetic condition of the sensory nerve fibrils in the wall. Patients now and then describe their stomach as giving them the idea of an absolutely inert mass or foreign body,—a symptom of nervous dyspepsia with temporary arrest of peristalsis and secretion, where the nerves of the surrounding abdominal viscera are hypersensitive. Sensations of

dragging down of the stomach are often very distressing; they appear in dilatation, in gastroptosis, where the pyloric end alone is displaced downwards—pyloroptosis,—frequently with floating kidney, and where peritonitic adhesions exist between the stomach and other abdominal organs or tissues.

The relationship between gastric uneasiness and painful sensations and the taking of food is an important factor in cases of indigestion. These symptoms may precede, accompany, or follow its ingestion. If coming after food the length of time which elapses before the morbid sensations set in is of value in determining their origin. When distress and pain occur at the time of entry of food into the stomach, if they be not more than slight though persistent, subacute or chronic catarrh of the gastric mucous membrane, malignant disease, dilatation with incomplete emptying and fermenting residual contents, or irritability of the nerve-endings in the mucosa, resenting the introduction of new material, may be present. More acute pain and discomfort coincident with entrance of food occur in gastric ulcer, in cases of cancer with ulceration, and in hyperæsthetic nervous cases.

The usual site of pain due to gastric disease is the epigastric region, extending up below the sternum in chronic gastritis, of a radiating and more continuous character in cancer of the pylorus, severe and strictly circumscribed in gastric ulcer, but associated with a similar small painful area in the back close to and usually a little to the left of the eleventh or twelfth dorsal vertebral spine; paroxysmal and localised in idiopathic gastralgia; dull and more diffuse in neurasthenia. Cancer of the smaller curvature of the stomach may give rise to painful sensations at a somewhat higher level, of the posterior wall to pain felt in the back. In dilatation and gastroptosis the location of the pain approaches nearer to the umbilicus. Gastralgia when caused reflexly by disease of other organs is usually located in the epigastric region, but the pain tends to spread over a wide area.

Cardialgia, a symptom commonly described as heartburn, proceeds from irritation of the nerve terminations in the lining membrane of the œsophagus and pharynx by hyperacid contents of the stomach, eructated by antiperistaltic action and initiated by over-stimulation of the gastric nerves by the excessive acidity of the contents. Heartburn is merely an extension of the less definite variety of pain induced by excess of acidity in the stomach itself to the œsophagus and pharynx, whose nerve-endings are more sensitive to acid stimuli.

2. *Motor Symptoms.*—The most frequent result of disturbance of gastric motility is delayed digestion, from weakened peristaltic movements of the muscular coat of the stomach wall, with some degree of stasis of the contents and fermentation. If severe or protracted, actual dilatation may follow. Hyperperistalsis is not so common, but is met with in irritable nervous cases, especially in those with hyperchlorhydria, and occasionally is so pronounced in character that the contractions are palpable. Pyloric spasmodic contractions are of a similar nature; irritation of the adjacent gastric surface by acid contents inducing the paroxysms which are frequently accompanied by severe cramp-like pains and burning sensations.

Vomiting or emesis is directly due to abnormal motor action, but is usually a secondary result of nerve irritation. Consisting in paroxysmal gastric antiperistalsis, combined with contraction of the diaphragmatic muscle and of the muscles of the abdominal parietes, it may be of local, remote, or central origin. If of local origin, the exciting cause may be in the stomach contents, or in the blood supplying the local nerve ganglia; if remote, the primary source is usually irritation of peripheral nerve fibres or cells, of the abdomen and pelvis in pregnancy and ovarian tumours, of the peritoneum in acute peritonitis and intestinal obstruction; and, if central, either of spinal or cerebral origin, from organic lesion, toxæmic irritation, or functional nervous disorder. Generally speaking, acts of vomiting which are independent of meals, are preceded by little nausea, and followed by few if any feelings of ill-health, and in which the materials vomited are of normal character, or nearly so, point to a functional cause, peripheral or central, uncomplicated by local organic disease, or by morbid changes of local function induced by neurotic influence. The relationship between the taking of food, the kind of food, the regularity or irregularity of occurrence, the degree of nausea experienced, the character and amount of the vomit, the features of the act itself, and an attack of emesis, requires investigation before the true significance of the symptom can be properly appreciated; in addition, of course, to the obtainal of positive or negative evidence as to the presence of complicating lesions, which may possibly be to blame.

Rumination or merycism is another motor symptom, and one indeed which is not so uncommon as popularly supposed. It may be, however, perfectly compatible with a healthy state of digestion, although tending to initiate symptoms of dyspeptic disturbance in course of time, if the habit be long and constantly persisted in. While met with in certain cases of indigestion, particularly in those of neurotic origin, it can scarcely be regarded as a true morbid symptom of dyspepsia, but rather as a more or less abnormal habit, often begun in curiosity, perceived to be not unpleasant, repeated too frequently, and ultimately found to have become a necessary custom. The habit has arisen from imitation, as in Körner's case,

and has been traced in a few instances to hereditary influence.

3. *Functional Signs and Symptoms.*—These denote the numerous errors of secretory and digestive power. The methods of examining the contents of stomach and the significance which attaches to their results have already been fully discussed in an earlier article. It will suffice here to introduce a scheme of classification indicative of the relationship between altered characters of the stomach contents and indigestion in many of its forms:—

Classification of Gastric Conditions with regard to the Characters of the Stomach Contents, Chemical and Physical

I. Acidity.
- A. Normal.
  In health; hepatic dyspepsia; cancer of cardiac orifice and œsophagus; erosions; nervous and anæmic dyspepsia.
- B. Hyperacidity.
  1. Hydrochloric acid increased.
     In gastric ulcer, and erosions; from nervous causes; in some cases of dilated stomach; occasionally in cancer following ulcer.
  2. Hydrochloric acid diminished; organic acids increased.
     In cancer of the pylorus and pyloric region; dilatation of the stomach; nervous dyspepsia; the later stages of gastric ulcer; chronic catarrh; atonic dyspepsia.
- C. Hyperacidity.
  1. Without organic acid fermentation.
     Hypochlorhydria without fermentation; at beginning of acute gastritis, or during convalescence from it; cancer of the stomach (rarely); cirrhosis ventriculi; during acute or chronic febrile and cachectic disorders (on occasion).
  2. With organic acid fermentation.
     Chronic catarrh; atrophy of the mucous membrane; cancer; waxy degeneration; cirrhosis; acute febrile diseases; dilated stomach without pronounced stasis; phlegmonous gastritis.

II. Digestive power.
- A. Normal.—In health; gastric ulcer; nervous dyspepsia; many cases of œsophageal and cardiac cancer; early catarrh.
- B. Increased. — Gastric ulcer; nervous hyperchlorhydria; often in diabetes.
- C. Diminished. — Cancer of stomach; chronic catarrh; atony; dilated stomach with catarrh; nervous dyspepsia; acute gastritis; acute fevers; cirrhosis.

III. Length of stay in stomach.
- A. Normal.
  In health; gastroptosis without obstruction; nervous dyspepsia; gastric ulcer.
- B. Shortened.
  Hyperchlorhydria; hyperperistalsis; insufficiency of pylorus; gastric ulcer (rarely), diabetes.
- C. Lengthened.
  1. Diminished peristalsis. Nervous dyspepsia; cancer; dilatation; pyloric stenosis; cicatrices of old ulcers.
  2. Decreased power of digestion. Cancer; chronic and atrophic catarrh; acute gastritis.
  3. From both causes. Cirrhosis of stomach; dilated stomach with catarrh; many nervous dyspepsias; hypersecretion (fluid remaining in form of gastric juice, not of food). Cancer at later stages.

IV. Specific gravity (after filtration).
1. Normal. After a meal 1010-1020.
2. Increased. Hyperacidity 1020 +
3. Diminished. Hyperacidity 1010 –

V. Blood.
1. Bright red, copious, coagulable. Gastric ulcer.
2. Darker, copious, clotted. Gastric ulcer or cancer; duodenal ulcer (rarely).
3. Bright red or brown, smaller amount. Gastric erosions, chronic ulcer, with weak cicatrix; acute gastritis; hepatic congestion; venous engorgement.
4. "Coffee grounds." Cancer, especially with atony and dilatation.

Special Forms of Indigestion

Hemicrania (Migraine, Megrim, Sick-headache).—The subject of megrim, its nature and significance, to be adequately discussed, would require a volume to itself. The question whether hemicranial attacks are species of nerve-tornadoes, as advanced by Liveing, are auto-toxæmic in origin, are caused by both of these factors in combination, or derived from various other suggested sources, boasts of so extensive a literature and has been so diversely answered that it is hopeless to go fully into it here. To the writer the most probable theory, and the one capable of the widest application, ascribes the exciting cause to a sudden overflow of abnormal nerve force, similar to that which occasions epileptic seizures, but expended upon sensory and functional nervous mechanisms in lieu of motor areas, incited thereto by the presence of poisonous substances in the blood, the products of insufficient metabolism, which, when they

attain to a certain proportion in the blood, initiate a critical paroxysm, followed by a longer or shorter period of quiescence. Megrim, associated with gouty habit, is explicable on similar grounds. When intimately connected in women with the menstrual function, the periodical stimulus to the nervous system thus afforded suffices to liberate any morbid surplus of nerve energy, in conjunction with the usual concomitants of such cases,—constipation and some degree of auto-intoxication. Long made a scapegoat in megrim, the liver is more probably a companion in misfortune than an organ meriting blame.

The symptoms accompanying attacks of megrim are most variable in character; practically the only features which are constantly presented being the paroxysmal nature of occurrence, the entire absence of cognate symptoms throughout the intervals, and the intense feeling of nausea produced during the later stages of the attack. Hemicrania; ocular disturbances, such as hemianopsia, scotoma, and subjective perception of apparent visual objects, coloured or uncoloured; mental confusion, paræsthetic symptoms on one side of the body, and post-paroxysmal nervous phenomena,—aphasia, temporary loss of memory, — may severally be present or absent.

Vomiting, or some other form of critical evacuation, usually intervenes before subsidence of an attack; but this is not invariably the case, the nausea sometimes disappearing gradually apart from actual crisis.

Flatulent Dyspepsia.—Gastric flatulence may be of two kinds: either the result of fermentative processes taking place in the contents of the stomach, or of nervous origin. When fermentation of food-stuffs constitutes the source of the gas there usually coexists a lack of hydrochloric acid, secretion of which has diminished owing to catarrh of the gastric mucous membrane, atrophy of its cells, or nervous inhibitory action; and the tendency towards gaseous evolution is increased in each instance if some degree of stagnation of the gastric contents is also present. Flatulence of this nature corresponds in degree and in character with the variety of food ingested; the more readily fermentescible the food-stuff the greater the ebulition of gas. Should the stomach be dilated, and residual remnants from former meals be left in it after the normal time of emptying, while the proportion of hydrochloric present is insufficient to prevent fermentation, the symptoms of flatulence may obtain irrespective of meal-time or form of food. The gas proceeding from fermentative processes generally possesses an odour corresponding to the nature of the process producing it; is most abundant after the ingestion of farinaceous foods; and is increased by the administration of alkalies. In rare instances the presence of marsh gas has been detected by its ignition on contact with a flame of the mouth.

In many examples of purely nervous dyspepsia, unassociated with fermentative processes, symptoms of flatulence are often met with, astonishing in the volume of gas evolved. Here the formation of gas is independent of the character and the ingestion of food; the gas itself is odourless, tasteless, is apparently expelled without effort or much discomfort, and may be due either to excessive entrance of carbonic acid gas from the blood stream into the stomach cavity, owing to abnormal nerve action, or to expulsion of atmospheric air previously swallowed. Indeed, air-swallowing and eructating forms a special type of neurotic indigestion.

Indigestion in Dilatation of the Stomach.—The degree of dilatation present modifies the character of the symptoms shown. In minor forms, where enlargement of the stomach scarcely amounts to true dilatation, the *megastria* of Ewald, characteristic symptoms may be absent, only a few of the signs common to chronic gastric catarrh of mild type attracting notice. Where the degree of dilatation is more pronounced, symptoms indicative of delayed emptying of the stomach-contents, of stasis, and fermentative processes in the residuum left in the viscus, appear in most instances. But it is of importance to remember that the symptoms presented by the possessors of a dilated stomach vary directly with the cause of the dilatation. Where the condition has arisen as the sequel of habitual over-distension of the organ with food or with fluid, leading ultimately to loss of muscular tone, the chief fault lies not so much in loss of digestive power as in defective motility; where dilatation follows atonic dyspepsia, after long-continued chronic catarrhal conditions have injured both the secretory and motor functions of the stomach, stasis of the contents is aggravated by deficient digestive properties; in the by no means infrequent examples of dilatation, to all appearance solely due to enfeebled resilience and motility through lack of efficient nervous control, a most incongruous series of symptoms may be presented, the ordinary symptoms of a dilated stomach being masked by some of the many possible in nervous dyspepsias; and, lastly, where dilatation of the stomach depends upon a stenosis of the pyloric orifice, the symptoms and signs correspond. In addition, it should be further mentioned that the signs accompanying gastric dilatation from pyloric stenosis vary with the nature of the constriction. Omitting consideration of cancer of the pylorus at present, simple hypertrophy of the pyloric muscular and connective-tissue coats may lead to narrowing of its lumen, and prove a hindrance to the escape of stomach contents, and contracture of cicatricial tissue over the site of an old ulcer acts similarly. Occasionally hyperacidity of the gastric contents leads to temporary spasmodic closure of the pyloric sphincter by reason of irritation of the nerve-

endings in the gastric mucous membrane; and, if frequently repeated, dilatation of the stomach may be a result, more especially where persistent irritation and consequent contracture has induced an actual hypertrophy of the pyloric tissue.

Analyses of the gastric contents in cases belonging to these several classes generally yield characteristic points of difference. Wherever stasis accompanied by loss of peptic power and deficiency of hydrochloric acid has taken place, examination of the contents affords evidence of an excess of organic acids of fermentative processes, and of decreased digestive activity. Should the contents be obtained from a dilated stomach of nervous origin, or following upon cicatricial pyloric obstruction, they may prove to contain hydrochloric acid in normal or excessive proportion.

Associated with Displacement of Abdominal Viscera.—This variety of indigestion is fully treated of elsewhere (Gastroptosis, Enteroptosis, Floating Kidney), and need not be further alluded to here, except to emphasise the possibility of dyspeptic symptoms arising from such lesions.

Œsophageal diverticula, congenital or acquired, may give rise to very pronounced symptoms of indigestion, attended with great difficulty in diagnosis. The special features shown by such cases chiefly consist in sudden acts of regurgitation of food, unassociated with nausea and the ordinary phenomena of emesis; the regurgitated material differing in composition from gastric contents, lacking hydrochloric acid and pepsin, although frequently of acid reaction from the presence of organic acids of fermentation, and as a rule including portions of food swallowed some time, some days even, beforehand.

Enough has already been said as to the factors which have a part in causing the appearance of dyspeptic symptoms, and whose origin must be sought for elsewhere, to indicate the necessity of careful investigation in all possible directions before a trustworthy diagnosis can be arrived at. This matter is so important that it may again be insisted on, that recognition of the probable existence of a primary cause, and appreciation of its true nature, are of more value in connection with indigestion than the greatest familiarity with the local symptoms common to it.

Treatment.—In few conditions is an intelligent and rational perception of the close relationship between cause and effect so valuable as in the treatment of indigestion. And, further, owing to the fact that the cognomen, indigestion, refers to symptoms of varied origin, although it may be similar in character, it is of the utmost importance to ascertain the nature of the true, as compared with the apparent, cause which produces the effect, before any satisfactory and efficient course of treatment is decided on.

Simple, passing attacks of mild indigestion, arising from brief and temporary errors of diet, of habit or circumstance, and characterised by the symptoms common to slight gastric catarrh, readily subside if a light diet be ordered, the exciting cause not repeated, and the bowels freely moved with one or other of the milder purgative drugs; if thought advisable, even if only as a "placebo," a gastric sedative may be given: one of the different forms in which bismuth is prepared, with or without alkaline carbonates. The main indication lies in stoppage of the direct source, light diet, never taken in large bulk at one time, and free evacuation of the bowel.

Acute cases of gastric catarrh, due to alcoholic excess, to severe local inflammation from irritating articles of food or toxins, or occurring during the course of subacute and chronic attacks, either from the principle of the summation of stimuli which in these cases accrue from long-continued dietetic irritation, or from ingestion of more than usually irritating material, benefit most from prompt emptying of the stomach, by giving an emetic, if indeed nature has not anticipated this, and from exhibition of a rapid purge, saline or cholagogue, to rid the intestine of toxic bodies. Milk and slops—no alcohol, tea, or coffee, no vegetables or fruits, cooked or raw—should form the diet, given every two or three hours during the day. Water, cold or hot,—aërated, with or without potash or soda, or mixed with the milk, — may be taken *ad libitum* as is found agreeable. The local gastric distress is best relieved by the application externally of heat or of a mustard plaster. The common coincidence of duodenal and acute gastric catarrh, leading to catarrhal jaundice, should be kept in mind, and the adoption of measures appropriate for the removal of this condition in addition to those directed against the gastritis will often prevent this sequela.

As few cases of mild indigestion of brief duration, or of acute gastritis, are due to more than a temporary inflammation of the gastric mucous membrane from irritating food-stuffs, or too large meals, the majority are best treated by regulation of diet and habit, and by giving the stomach a period of comparative rest; the less medicine used the better.

If, however, such attacks are accompaniments of more general morbid conditions, such as anæmia, chlorosis, gout, valvular heart disease, portal obstruction, and neurasthenia, it is not sufficient to treat them alone apart altogether from the really more important general lesion. In truth, under these circumstances it is more difficult to treat them successfully at the time, while they display an immensely greater tendency to recur.

But it is in the chronic and protracted sub-

acute types of indigestion that the question, Which line of treatment is proper for adoption? comes to be hardest to answer—that the importance of recognising the nature of the primary source of the evil ranks highest. For here vicious circles have usually been established. Recurring attacks of simple or acute gastric catarrh have led to chronic inflammatory changes; deficient peptic digestion to intestinal disturbance; diminished absorption of nutriment to metabolic error, to anæmia, gout, inertia, and nervous debility; or *mutatis mutandis* anæmia, gout, reflex influences, and neurasthenia have induced dyspeptic symptoms. Whether the indigestion precede the other conditions, or *per contra* arise from them, their coexistence aggravates both forms of disease, and direct treatment of the one is often injurious to the other.

The problem then to be solved is whether it would be best to deal first with the dyspeptic condition or with the more general error. The answer depends largely upon the presence or absence of organic lesion in the gastric mucosa, upon whether the processes of digestion are actually amiss, or only subjectively annoying; whether the general or the local condition is the more important; and upon the greater likelihood of benefiting the one by preliminary attention to the other than *vice versa.*

Alkalies given before meals tend to increase the acidity of the gastric contents after them; dilute mineral acids, too, act conversely. Given with or after food they exactly reverse their actions. The effect, however, of alkalies given with or after food in lowering the acidity of the stomach contents is purely equational, and exactly corresponds in degree and duration to the amount introduced. If sufficient time elapses before emptying of the stomach the decrease may become increase, the presence of the alkali inciting an enhanced secretion of hydrochloric acid. As the secretion of this acid is governed by the force of the exciting stimulus, and displays an intimate connection with the degree and character of gastric acidity present, the introduction of alkalies into the stomach calls for greater secretion of acid, of acids or less.

Alkaline drugs, therefore, given to quell acidity in reality serve ultimately to heighten it; acids recommended for hypo-acidity are liable to weaken in place of strengthening the already deficient acid secretion. These statements only apply when disturbing complications are absent.

The therapeutic value of the digestive ferments is a disputed matter. Predigested food often proves of great if temporary service; but the exhibition of pepsin, pancreatin, etc., by the mouth most probably exerts less beneficial effect in gastric errors than has been ascribed to it by some.

Dilute hydrocyanic acid in small doses frequently repeated, with or without some form of bismuth, often suffices to soothe gastric pain; now and then, if severe in character, morphina or codeina may be required.

Instrumental Measures.—Lavage is undoubtedly of service in certain cases, but its beneficial properties have probably been much exaggerated, and certainly its practice has often been abused. Lavage may be imperative in pronounced dilatation of the stomach, or excessive pyloric stenosis; in other cases the influence of contemporary treatment, medicinal and dietetic, and the effect upon the psychical centres exerted by the details of the process, require to be eliminated. Where removal of the stomach contents from time to time is not obligatory by reason of some absolute hindrance to their downward passage, lavage if habitual is unnatural, illogical, betokening absence of resource on the part of the physician, or the induction of a habit contributed to by him.

Electricity applied to the surface of the stomach itself, externally over the gastric region, or over the segments of the spinal cord related to it, sometimes produces very beneficial results; but the effects produced by it are of so variable a nature, that actual trial alone can suffice to prove whether it may be of service or not in any particular case. Further allusion to the merits or demerits of these, and of the many other instrumental means of intragastric treatment which have been devised, goes beyond the scope of this article.

Diet.—It is as important to base the choice of a suitable diet upon the nature of each individual case, as it is proper to modify the lines of medicinal treatment upon the same. Too frequently some particular form of diet comes to be recommended as a routine practice for all dyspepsias, whatsoever be the type. The favourite diet may vary from time to time, seldom from case to case. Personally the writer has little faith in, and considerable distrust of, the imposition of strict, rigid rules for diet, of restriction within narrow limits as to food, definite, or what is still worse, indefinite in duration. In simple transient catarrh, or in cases of acute gastritis, brief limitation to the simplest food proves excellent. In the majority of the more prolonged cases of indigestion, and these are the cases which most often call for medical treatment, a longer or shorter course of restriction to a few specified food articles frequently takes the form of punishment, and defeats its own object by arousing feelings of repulsion through the constant sameness of the viands permitted, and of depression consequent thereon. Monotony deadens enjoyment, weakens appetite, injures digestive power, while increasing morbid nerve influence.

It were more rational to be guided by signs shown by nature, and to endeavour to facilitate the progress towards recovery along natural lines,

than to institute for a considerable period of time immutable and cribbed conditions, disregarding any changes which may occur intercurrently—conditions as unlike the salutary variations always exhibited in nature's doings as is possible.

How far psychical changes in temperament represent concrete alterations of substance in nerve ganglia, or differences in the processes productive of nerve force, is as yet undecided; be the relation what it may, psychical temperament exerts marked influence over functional activity throughout the body, and concerns our methods of therapy. All that tends to depress the mind tends to diminish functional activity, and to intensify or prolong existing morbid conditions. The dietetic rule, which, while benefiting the more local errors of digestion, is best calculated to stimulate the psychical functions, and not to cause mental depression, which progresses in variety of available foods as the condition of the patient improves, while strictly remaining within the limits of tolerance, is the rule, the adoption of which is likeliest to be followed by satisfactory results, general as well as local, and lasting in effect.

Gastric processes are principally concerned in the digestion of proteids, in the disintegration of masses of flesh, and in the thorough intermixture of all the food-stuffs taken. Normally it has little or no action on carbohydrates or fats, except by reason of its acidity in preventing their fermentation. Wherever the secretion of hydrochloric acid is decreased, and, therefore, abnormal fermentative changes in carbohydrate substances facilitated, farinaceous foods should be largely forbidden, especially those which are most readily fermentescible. The diet given, varied according to circumstance, should be mainly composed of the blandest forms of proteid foods, never taken in large quantity at any one time. The normal powers of digestion possessed by the stomach juice are lessened; they should, therefore, be given an easier task to do, while guarded as far as possible from further irritation.

Indeed, it almost seems to be quite rational to assume that whenever the stomach is sick the diet should mainly consist of that class of food-stuffs which is naturally acted upon by its secretions, discarding the foods which are almost as foreign bodies to it.

Nervous Dyspepsia.—The multitudinous manifestations of indigestion proceeding from nervous causes require both local and general treatment. Here, again, the subject is complicated by the question of *post hoc, vel propter hoc.* Are the symptoms the result of morbid nervous affection, or have the dyspeptic lesions brought on nervous disorder? While, again, the possibility of a purely reflex causation of the symptoms of indigestion adds to the difficulty.

As a rule local treatment can only be palliative, although in so far as it benefits the general nutrition of the body it helps towards ultimate cure; while removal of the source of the nervous disturbance is the real goal to be aimed at. If the symptoms arise reflexly from local disease in other regions, as is frequently the case, relief will follow most surely from the cure or improvement of the remote causal agency. Diseased conditions of the large bowel and rectum, of the female pelvic organs, displacements of other abdominal viscera than the stomach, pregnancy, and disordered menstruation, may require attention. In several instances the writer has procured rapid relief of dyspeptic symptoms in women at or past the menopause, or who had undergone double ovariotomy, by the sole employment of ovarian extract or didymin—all local calls for treatment being entirely ignored. When the underlying cause consists in nervous debility, neurasthenia, or nervous irritability, choice of correct therapeutic measures is a matter for grave consideration. Treatment of symptoms in accordance with the nature of the local lesions inducing them, as revealed by analytical examinations of the gastric contents, or in the light of previous experience of like cases, prohibition of dietetic transgressions, and prescription of nerve sedatives may help. Far more benefit follows change of habit, of environment, and of interest; less (or more) professional work or manual labour; less (or more) physical and mental exercise; less (or more) opportunity for careful regard to personal traits, phenomena, and wants. Many of the victims of nervous dyspepsia, who have developed hypochondriacal tendencies, derive special benefit from change of scene and circumstance, with absence from routine work, and diversion of thought. Others, again, seem to experience a larger degree of relief from assiduous local therapy, especially if employment of physical methods be associated with it.

Excessive emaciation in neurasthenic subjects, with neurotic dyspepsia, especially that type known as *anorexia nervosa*, can only be successfully treated away from home and by some form of the treatment associated with the name of Weir-Mitchell, into which rest, massage, and a generous supply of simple food enter.

In Anæmia and Chlorosis.—In treating the symptoms of chronic indigestion so often met with in anæmic and chlorotic subjects the possibility of gastric ulcer should always be borne in mind,—acute, in course of formation, and existing in part, or chronic, imperfectly healed, or cicatrised. The increased sensitiveness of the nerve-endings in the gastric mucous membrane towards the irritant effects of free acid in the contents, because of the lowered nutritive properties of the blood supplied to them, must also be taken into account. Their digestive powers may be normal, along with severe symptoms of gastric disorder, arising solely from the anæmic state of the blood.

When the symptoms observed point to irritative dyspepsia, and the gastric contents contain a normal proportion of free hydrochloric acid, or even a somewhat diminished amount, treatment should be directed towards mitigation of the irritant effects of the free acid and improvement of the state of the blood. In such cases many of the ferrous salts are well tolerated.

It is a much more troublesome matter to treat successfully anæmia complicated by atonic and hypo-acid indigestion, for hæmatinic drugs are ill borne by the stomach, while measures devised for removal of the gastric condition are often prone to harm rather than benefit the anæmia. The most rational method to adopt, perhaps, is to endeavour, in the first place, to improve the powers of gastric digestion, giving at the same time such non-irritating tonic drugs, not necessarily ferruginous, as strychnine, phosphoric acid, etc., and then combining with the gastric measures, when the stomach has recovered much of its tone, one of the least astringent preparations of iron—selection of the individual form being made in each case after careful consideration of all the surrounding circumstances, and guided by the results of actual trial.

Dilated Stomach.—As the symptoms of indigestion accompanying dilatation of the stomach are mainly those of delayed emptying of that viscus, with consequent stasis of and subsequent onset of fermentative processes in its contents, treatment must be directed towards removal or relief of such aberration of function, but must also take cognisance of the primary cause which has led to the dilatation, whether it is the sequel to pyloric obstruction, organic or spasmodic; be the result of myasthenia gastrica, with loss of resiliency of the muscular coat; of habitual over-filling of the organ throughout a prolonged period of time; of frequent overdistension in flatulent dyspepsia; or, again, the after-effect of repeated and protracted attacks of chronic catarrh. Little can be done to relieve the symptoms which are due to organic pyloric stenosis, save by regular removal of the stomach contents with the stomach tube, after every chance has been given for the absorption of all available nutriment through the stomach mucous membrane on each occasion; or by operative interference and excision of the constricted pylorus with formation of a gastro-duodenal intercommunication. Spasmodic closure of the pyloric sphincter is commonly the result of irritation from hyperacid contents of neurotic origin; and the gastric dilatation which sometimes follows repeated attacks of this form of contracture, may readily disappear on removal of the exciting cause, provided that hypertrophy of the pyloric tissues has not been induced to any great extent.

The treatment of *gouty indigestion*, particularly where the indigestion belongs to the chronic nervous variety, is fraught with many difficulties. The gastric and intestinal symptoms can rarely be treated save to the detriment of the gout; the gout, but with harm to the dyspepsia. In some it may appear advisable to deal directly with the digestive symptoms first; in others to influence them indirectly through the alleviation of the gouty habit. If the type of indigestion present permit, fractional dieting, mostly by milk, with copious flushings out of the organism with large amounts of plain water, or non-irritating mineral waters, such as Contrexéville, may benefit both factors.

Indigo. *See* Pigments of the Body and Excreta (*Indigo Pigments*); Toxicology (*Arsenic*); Urine, Pathological Changes in (*Calculi, Indigo*).

Indigouria.—The presence of indigo in the urine, due to decomposition of indican.

Indol.—Amido-ethyl-benzene. *See* Fæces (*Odour, Aromatic Substances*); Physiology, Food and Digestion (*Bacterial Action in the Alimentary Canal*); Physiology, Excretion (*Urine, Sulphur-containing Bodies*).

Indoxyl. *See* Physiology, Excretion (*Urine, Sulphur-containing Bodies*); Urine, Pathological Changes in (*Aromatic Substances*).

Indoxyluria.—The presence of indoxyl (*q.v.*) in the urine.

Induced Electricity. *See* Physiology, Tissues (*Muscle, Methods of causing Contraction*).

Induction of Labour. *See* Labour, Operations (*Premature Induction of Labour*).

Induration.—An increase in the consistence of an organ or tissue due to the growth in it of tissues harder than that of which it is normally composed, *e.g.* fibrous tissue; it may be caused by prolonged venous congestion.

Industrial Diseases. *See* Trades, Dangerous; Toxicology.

Inebriate Act. *See* Alcoholism (*Treatment, Social*).

Inebriety. *See* Alcoholism; Vice.

Inertia.—Atony or diminished power of action, *e.g.* inertia uteri, when in labour that organ has its power of contraction lessened, and mental inertia in which there is sluggishness of mental action.

Infancy and Infant. *See* Atrophy, Infantile; Children, Development of; Children, Clinical Examination of; Cholera Nostras; Climate (*Hot, Infantile Mortality*);

Cretinism; Deafmutism; Deformities (*Infantile Hemiplegia, etc.*); Deformities (*Club Foot*); Diet; Gastro-Intestinal Disorders of Infancy; Hæmoglobinuria (*Infantile*); Hip-Joint, Diseases of (*Morbus Coxæ, Diagnosis*); Hysteria in Childhood; Hernia; Homo Caudatus; Infant Feeding; Invalid Feeding; Knee-Joint, Diseases of (*Infantile Paralysis*); Labour; Larynx, Congenital Laryngeal Stridor; Larynx, Laryngismus Stridulus; Medicine, Forensic (*Infanticide*); Mental Deficiency; Muscles, Diseases of (*Idiopathic Muscular Atrophy, Infantile Type*); New-Born Infant; Nursery Hygiene; Paralysis (*Infantile Spastic*); Paralysis (*Infantile Hemiplegia*); Pulse (*in Infancy*); Sclerotic, Diseases of the (*Staphyloma*); Scrotum and Testicle, Diseases of the (*Orchitis, Hydrocele*); Teratology; Thymus Gland (*Enlargement*); Urticaria (*Symptoms*); Vital Statistics (*Infant Mortality*).

Infant Feeding.

The importance of an accurate knowledge of the principles and practice of infant feeding can hardly be overestimated. The appalling mortality of infancy is due in no small degree either directly or indirectly to faulty feeding. Nor is this faultiness of feeding limited to the poorer classes, it is by no means uncommon amongst the wealthy, and it would seem to be due more often to ignorance than to lack of means.

Of recent years much has been written concerning the physiology and chemistry of infant feeding, and there is a growing tendency in practice also to transfer the feeding of infants from the old rule of thumb to the region of exact science. In the present article the physiological and chemical aspects of the subject will be considered chiefly in their direct bearing upon the practical problems of infant feeding, but it may be well to make some preliminary remarks with reference to these points.

In a general way it may be said that infants require the same food-constituents as adults. To repair the waste of tissue metabolism at any period of life the food must contain five elements, namely, proteids, hydrocarbons, carbohydrates, salts, and water. But the most elementary knowledge of the physiology of infancy shows that these constituents must be supplied in very different forms and proportions from those which can be used for adults or even for children over the age of two years.

During the first six months of life the teeth have usually not yet appeared, no food therefore which requires mastication, such as bread, potatoes, or puddings, can be suitable at that age, indeed in many cases, even at the age of twelve months, the teeth are not sufficiently advanced to deal with such articles of diet.

The saliva and pancreatic secretion only gradually acquire their full amylolytic power. The writer found that distinct though feeble amylolytic action was exerted by an infusion of the parotid gland from an infant still-born at full term; it would seem, however, that the saliva does not acquire any considerable degree of this power until the infant is three months old, while the pancreatic secretion which plays the most important part in the digestion of starch does not attain to its full amylolytic power until several months later, perhaps not until the end of the tenth or eleventh month. For this reason the carbohydrate element in food must not be supplied in the form of starch during the earlier months of life, it must be given in the form of sugar. The difficulty in digesting such articles as bread, potatoes, or any of the numerous so-called "infant-foods" which contain starch, can therefore readily be understood. The gastric juice which plays the chief part in the digestion of proteids has its peptonising powers well developed from the time of birth, so physiologists tell us; but experience shows that these powers are but feeble compared with those in older children, and that any but the most easily digestible of proteids quickly overtax the infant's stomach. This is particularly noticeable in the difficulty which infants find in digesting the casein in cow's milk. But here another peculiarity of early life has to be considered. The thinness and weakness of the muscular wall of the stomach and its feeble contractions render peristalsis less effective than in later life, so that solid masses, such as are formed by the casein of cow's milk, may be less thoroughly mixed with the gastric secretion, and therefore less easily digested.

But there are other considerations which determine to some extent the diet of infancy. Metabolism at this period is altogether more rapid than in later years; infancy is *par excellence* the age of construction. Never again does anything occur comparable to the extraordinary rapidity of growth in infancy. That an infant

should actually double its weight in five months and treble it in twelve is evidence of the marvellous activity of tissue growth at this period. To supply material sufficiently rapidly, not merely to compensate for the waste which accompanies metabolism, but also to serve for the building of the growing structure, it is necessary to feed much more frequently than in later life, and to give the food in such a form that it shall be capable of ready absorption and assimilation. The most perfect fulfilment of these requirements is seen in the mother's milk, with its finely divided curd, so easily acted upon by the gastric juice, its comparatively large proportion of soluble proteid, and still larger proportion of soluble carbohydrate.

The importance of a large proportion of fat in the diet corresponds to the vital necessity for warmth at this age. To supply this warmth the infant requires a considerably larger proportion of hydrocarbon, the heat-producing element in the food, than adults do. Physiological observations have shown that while for an adult the proportion of fat to proteid required is about 1 to 3, for infants it is nearly 2 to 1. But the need of warmth is certainly not the only reason why more fat is required for infants than for adults. It would seem that fat, taken as such in the food, plays an important part, directly or indirectly, in the growth of the tissues; in particular there is strong evidence to show that if the food be deficient in fat the growing bone is very apt to show rickety deformities.

Human Milk

The only food which fulfils all the requirements of early infancy is human milk, and the nearer any other food approximates in its character to human milk the more suitable it is for the feeding of infants.

Human milk is an opaque, yellowish-white fluid, with an average specific gravity of 1030-1032 (the writer found variations from 1027 to 1034). It is neutral or faintly alkaline in reaction. Its taste is sweet. It is usually sterile, but recent investigation has shown that this is not always so, the staphylococcus aureus and albus are sometimes found in it, and it seems probable that these and other bacteria may find their way into the ducts from the outside.

Microscopic examination shows that the milk consists of two parts: (i.) A colourless plasma, which contains proteids and salts in solution; (ii.) Globules of fat suspended in the plasma. The proteids found dissolved in the plasma are chiefly of two kinds: (1) Casein, or more accurately speaking, caseinogen, which is precipitated by acids, and also by a special ferment, rennet, in the gastric juice; (2) Lactalbumen, which is not precipitated either by acids or by rennet, but is coagulated by boiling. The fat globules in human milk measure from $\frac{1}{3000}$ to $\frac{1}{12,000}$ inch in diameter.

Many analyses of human milk have been made with widely-varying results. The practical difficulty is great, for unless the whole contents of the breast are used no strictly accurate result can be obtained, and it is not easy to secure the whole of the milk. The discrepancies between various observations are no doubt due partly to this fact, for the composition of the milk varies according to the part taken for examination. The milk may be divided into three portions, the fore-milk, the middle-milk, and the after-milk or strippings. An examination of the fat alone will show how widely these may differ in composition. In a specimen of fore-milk the writer found 1·4 per cent of fat, in one of after-milk 10 per cent of fat; in one case the fore-milk showed only ·4 per cent of fat, while the middle-milk showed 3·2 per cent. The proportion of proteid, similarly, is found to increase gradually, being lowest in the fore-milk and highest in the after-milk. It is obvious, therefore, that to obtain the average proportions of the various constituents a mixed sample of the whole contents of the breast should be taken. In practice this is seldom possible, and the next best course is to use only the middle-milk, from which presumably a fair average estimate can be obtained. A convenient method is to inquire how long the infant usually takes to empty the breast, and then to direct that it shall be put to the breast for one-third of this time, usually about five minutes, after which the specimen of milk, which should not be less than one ounce, is drawn off for analysis.

The average composition of human milk, calculated from various published analyses, may be stated as follows:—

	Per cent.	Variations.
Proteids— Casein, ·6; Lactalbumen, 1·4	2·0	(1-4·8)
Fat	3·5	(2·4-4·2)
Lactose	7·0	(5·8-7·4)
Salts	·2	(·1-·37)
Water	87·3	(86·7-88·5)

Colostrum.—During the first two or three days after parturition, before the flow of milk is established, the mother's breast yields a fluid, colostrum, which has a somewhat different composition. It contains less fat and sugar, but a larger proportion of proteid. Microscopically, also, it shows certain large, round, or ovoid granular bodies, the "colostrum corpuscles," which are three or four times as large as the fat globules in later milk. In addition to its food-value the colostrum has been thought to have some laxative effect; this is certainly not due to excess of fat; in ten samples examined by the writer the average percentage of fat was 2·4, and some contained only 1·0 or 1·2 per cent. The curd formed with an acid was in some of these cases much firmer and larger than is usually found in ordinary human milk.

Breast-Feeding

Importance of Breast-Feeding.—No artificial infant feeding can ever be so satisfactory as nature's method of suckling at the mother's breast. One can hardly condemn too strongly the selfishness of those women who refuse to feed their infants in the natural way because suckling interferes with their social pleasures or engagements. From some statistics collected by the writer at the Hospital for Sick Children, it would seem that 90 per cent of the fatal cases of diarrhœa in infancy occur in those who are being partially or entirely hand-fed, only 10 per cent are in infants fed entirely at the breast.

Artificial feeding is responsible for the vast majority of the cases of gastro-intestinal disorder in infancy, while infantile scurvy and rickets are pre-eminently diseases produced by artificial diet, and that too in many cases where considerable care has been exercised in the feeding of the infant. Even where a scanty secretion of milk or other circumstances render it impossible for the mother to feed the child entirely at the breast, it is most important that she should continue partial breast-feeding, for it has been shown that even partial suckling considerably diminishes the risk of fatal diarrhœa, and also the liability to other disorders which are due to artificial feeding.

Conditions which contra-indicate Breast-Feeding.—In many cases arrest of the milk secretion necessitates feeding by hand, but there are other conditions which may make it advisable to wean the infant.

General debility in the mother is sometimes a sufficient reason for weaning the child; too often, however, this is merely an excuse for getting out of a disagreeable duty, and because a mother says she "never could suckle her children," it by no means follows that she cannot suckle her present infant; the medical man must judge for himself whether the general ill-health is sufficient to justify partial or complete weaning of the infant.

It occasionally happens that the mother's milk persistently disagrees with the infant. The reasons for this will be considered below; here it need only be pointed out, that in many cases by proper regulation of the mother's habits and diet it is possible to correct the fault in the milk, and that until such an effort has been made the child should not be weaned.

Tuberculosis in the mother makes it unadvisable for her to suckle her infant. If one may judge from the experimental evidence as to tuberculous cows the risk of infection is extremely small, unless the breast itself is tuberculous; but so long as there is any active tuberculosis in the body the possibility of infection by the milk cannot be absolutely excluded, and, moreover, the activity of the disease in the mother seems in some cases to be increased by suckling, so that it is wiser on the whole to wean. The presence of old tuberculous lesions, such as spinal curvature or scars of tuberculous glands, so long as they are quiescent and there is no active focus of infection elsewhere, need not prevent the mother from suckling her infant.

Syphilis in the mother does not *per se* contra-indicate suckling, except in the very rare cases where syphilis is acquired during pregnancy and the child shows no evidence of syphilitic taint. Where the mother shows no symptoms of syphilis, but the infant shows evidence of the inherited disease, there is some doubt as to whether suckling should be allowed. Apparent exceptions to Colles's law have occurred, in which the infant has infected the mother in this way, but such cases are extremely rare, and the vital importance of breast-feeding, especially for the weakly syphilitic infant, has to be balanced against the very remote chance of infecting the mother.

The risk of infection is probably much greater for a wet-nurse, who certainly should never be allowed to nurse such an infant.

Infants have occasionally acquired syphilis after birth; the risk of infection from such infants is great, and necessitates immediate weaning.

The occurrence of an acute febrile disease on the mother, if it last more than two or three days, may necessitate taking the child from the breast, partly because the milk under these conditions sometimes disagrees with the child, and partly for the mother's sake; but it is well to remember that even after the child has been kept from the breast for two, three, or even four weeks, it is sometimes possible to start the flow of milk again by putting the child back to the breast, and in some cases the secretion will continue if the milk is drawn off by a breast-pump regularly during the mother's illness. Often, however, the milk ceases altogether if suckling is stopped, even for a few days; it is important, therefore, not to take the child from the breast unless it becomes absolutely necessary. Puerperal fever or any other of the infectious fevers necessitate weaning.

The return of the menses during lactation is no reason in itself for weaning. An idea seems to be prevalent that when this occurs the milk necessarily becomes harmful to the infant. Undoubtedly the milk does sometimes disagree with the infant during the menstrual period, but even where this occurs the disturbance is usually only transitory and slight, and more often there is no disturbance whatever. That some variation may occur in the composition of the milk during menstruation has been shown by analysis, in some cases the proteids and fat have been increased, in others diminished; but in most cases the variation is so slight as to produce no evil effect.

Pregnancy occurring during lactation does not necessarily call for immediate weaning. It seldom happens that pregnancy occurs before the infant is some months old, and lactation can usually be prolonged with safety for three or four months after the mother becomes pregnant; it is generally wiser not to continue the suckling longer than this, especially where the mother has had previous miscarriages or is in feeble health. If the quality of the milk seems to be poor the infant should be weaned. Dr. Cheadle states that the only case in which he has seen rickets occur in an infant at the breast was one in which pregnancy occurred during lactation.

Failure of nutrition or symptoms of gastro-intestinal disturbance in the infant are considered last in this connection, for it is rarely indeed that they contra-indicate breast-feeding. One of the commonest of complaints is that the mother's milk "does not seem to satisfy the child," and mothers sometimes suggest weaning, or more often the addition of artificial food, on this account. Such a complaint should never be considered adequate reason for premature weaning. In many such cases the apparent hunger of the infant is really the result of dyspepsia from too frequent, too greedy, or too irregular feeding, and the child becomes more content when the breast-feeding is properly regulated; in other cases the milk is scanty, but by judicious dieting of the mother it may be possible to increase the supply; in others again, but these more rare, the composition of the mother's milk is at fault, and if it cannot be altered by proper regulation of the mother's habits it may be necessary to wean the infant. These points will be considered more fully below, here it will be sufficient to say that even partial artificial feeding is not to be recommended unless absolutely necessary, and in all such cases the surest guide is the progress of the infant's weight; complete weaning is only very rarely to be recommended in such cases.

Directions for Breast-Feeding.—An infant should be put to the breast within three or four hours of birth; by the mere action of sucking it stimulates the mother's uterus to contract, and the possible laxative action of the colostrum apart from its nutritive value may be beneficial. As a rule, for the first two or three days the very small amount of colostrum which is obtainable is quite sufficient, but when the infant is very feeble it is sometimes advisable to give it other food; for this purpose whey may be given from a spoon (a dessert-spoonful or tablespoonful every three or four hours) until the secretion of milk is established. Infants will sometimes refuse the breast altogether owing to the mother's awkwardness in giving it; it may therefore be necessary to explain the manner of nursing, especially to a young mother. The child should be held on the arm of the same side as the breast which is to be used, in a comfortable semi-recumbent position, with the head slightly raised and the body tilted slightly towards the mother, who should bend slightly forward. The nipple should be supported between the index and the middle fingers of the disengaged hand, which can thus be used either to control the flow of milk by squeezing the nipple between the fingers, or to assist the flow by compressing the breast with the palm of the hand.

Depressed nipples may give considerable trouble. The putting of the infant to both breasts shortly after birth while they are still flaccid before the milk secretion begins, allows it to grasp the nipple and pull it out more easily than it could when the breast is tense; but in many cases artificial suction is necessary, and if this fail, and the depression is such as to prevent the child from sucking, it is worth while to try whether sucking can be accomplished through a rubber nipple attached to a glass breast-shield. As a last resource the milk may be drawn off by a breast pump, and given to the child in a spoon or bottle as long as the milk can be obtained thus.

The breasts should be used alternately; one breast contains, as a rule, sufficient milk for one meal. The infant should take about fifteen minutes to empty the breast; it should never be allowed to fall asleep with the nipple in its mouth. It should be accustomed from the beginning to lie quietly in its cot after feeding: many infants will vomit if jolted in the mother's arms just after a meal.

The nipple after feeding should be washed with warm water and carefully dried.

Frequency and Regularity of Breast-Feeding.—During the first two days after birth, before the milk secretion is established, the infant should be put to the breast every four hours. After this the infant should be fed as follows:—

	Interval between Meals.
To end of first month	2 hours
To end of third month	$2\frac{1}{2}$,,
Until weaning	3 ,,

Even from the beginning it is important to accustom the child to miss one feed at night, so that a period of at least four hours' uninterrupted rest may be ensured to the mother. This interval without food can gradually be increased, until at the age of three months the infant will sleep as long as six hours at night, the last feed being given at 11 P.M., and the first morning feed at 5 A.M. After the age of six months a healthy infant may be allowed to sleep for as long as seven or even eight hours at night if it will, without being disturbed for food.

One is sometimes asked the question, whether an infant should be disturbed during the day if asleep at the time when its feed is due. Undoubtedly it should: regularity in the time of

feeding is most important both for the mother's sake and for the child's, and the disturbance of sleep is of little consequence, as an infant usually falls asleep again very quickly after it has been fed. It is quite easy to accustom an infant to expect its meals at regular intervals as timed by the clock; indeed, some infants will wake almost to the minute when their feed is due, if only these regular habits are insisted upon from the very beginning. Unfortunately the habit is easily broken, and on this account the rule of regularity in infant feeding must be as the laws of the Medes and Persians. Some of the commonest troubles of infancy—such as flatulence, colic, hiccough, fretfulness and sleeplessness—are due to neglect of this rule.

Some writers have advocated the use of one artificial feed daily, on the ground that circumstances may occasionally make it impossible for the mother to suckle at the proper time, and that if the infant is accustomed to take food from the spoon or bottle a difficulty is thus avoided, and also that it facilitates weaning. In the present writer's opinion such artificial feeding as a routine practice is certainly not to be recommended: it encourages the mother to think lightly of the importance of breast-feeding, in fact it facilitates neglect of her duty; it introduces a very serious element of risk which is totally unnecessary; and, lastly, any real difficulty in weaning is so exceptional that it may be disregarded.

Variations in Human Milk

Quantity.—According to some observations quoted by Dr. Holt, it would seem that the average quantity of milk secreted in the twenty-four hours increases gradually during lactation from 10-16 oz. at the end of the first week, up to 30-40 oz. at the sixth to ninth month. The amount taken by the infant at each feed also increases from $\frac{5}{8}$ to $1\frac{1}{2}$ oz. during the first week up to about 3 oz. at the end of the first month, and then varies from 2 oz. to 5 oz. at the end of the third month, and from 3 oz. to 7 oz. from the third month onwards. The accurate measurement of the milk secreted by the mother, and also of that taken by the infant, is no easy matter. The latter may be determined roughly by weighing the infant immediately before and after each meal; the total amount calculated thus for the twenty-four hours gives approximately the quantity secreted in that time by the mother. A more direct and accurate method of determining the daily secretion would be to draw off the contents of the breasts at intervals during the day and measure them, but this is hardly ever feasible. Fortunately for practical purposes, it is very rarely necessary to make any actual measurement of the milk, for any marked deficiency can hardly escape the mother's observation, and is sometimes obvious to any one who watches the infant at the breast—it sucks vigorously for a few minutes, and then failing to obtain milk, rejects the nipple and cries out peevishly; in other cases it remains sucking much longer than it should, perhaps as long as half an hour or more, evidently getting the milk very slowly.

Deficiency of Milk.—Even if there is reason to think that the quantity of milk secreted is below the average, it must not be hastily concluded that the addition of artificial food is necessary. The quantity may be quite sufficient for the needs of the infant, and the most reliable criterion of this sufficiency, as already pointed out, is the weight of the infant; so long as this is increasing it is not only unnecessary but unwise to make any addition. Where the breast milk is insufficient it may be increased by the mother's taking milk and gruel freely. In some cases the same result may be obtained by taking malt extract.

Malted liquors certainly increase the flow, and some mothers are enabled to feed their infants by taking half a pint of good beer or stout daily; this is, however, quite unnecessary as a rule, and should never be ordered without special reason, for apart from other obvious objections, the drinking of such a beverage by the mother sometimes alters the character of the milk so that it disagrees with the infant.

Excess of milk is quite uncommon, and but rarely calls for treatment. The fluids in the mother's diet should be restricted, and if there is any sign of the breast becoming unduly hard or tender, some of the milk should be drawn off two or three times a day in the intervals between the infant's feeds.

Quality.—The proportion of each constituent of human milk is subject to some variation, and this is noticeable even in samples taken from the same mother at short intervals. The sugar and salts would seem to be more constant than the others, and their slight variations are of no practical importance. The fat and proteids show considerable variations which are of great importance, for excess or deficiency of either of these constituents may render the milk unsuitable for the infant.

The actual proportions of each constituent can only be determined by analysis. Unfortunately there is no simple method available for clinical purposes in the estimation of the proteids, but a rough estimate can often be made from the specific gravity if the proportion of fat is known. The sugar and salts may be considered as practically constant, and therefore any variation in specific gravity depends upon the fats or the proteids. The higher the proportion of fat the lower is the specific gravity, and consequently, if with a high proportion of fat the specific gravity is high the proportion of proteids must be above the average; if with a low proportion of fat the specific gravity is low, the proportion of proteids must be below the average.

If the proportion of fat is normal, then the specific gravity will vary directly as the proportion of proteids.

For determining the specific gravity of breast milk it is convenient to use glass beads specially made to float in fluids of certain specific gravity; in this way, with an ordinary test-tube or small conical medicine glass it is possible to determine the specific gravity where only a few drachms of breast milk can be obtained. It may be repeated here that only the middle-milk must be used for any such estimation of the quality of the milk, for as mentioned above, an entirely erroneous estimate will be obtained if either the fore-milk or the after-milk is used.

The fat may be estimated by the following simple method :—A cylindrical five-cubic centimetre measure, having a diameter of $\frac{3}{8}$ inch, and graduated in cubic centimetres and millimetres, is filled with the breast milk exactly up to the five-cubic centimetre mark ; it is then corked and allowed to stand in a warm room (65° F.) for twenty-four hours. The cream rises to the top and is read off in cubic millimetres. Empirically the cream readings are found to correspond with fat percentages approximately thus :—

Cream.		Fat.
2 cub. millim.	=	2·8 per cent
3 ,, ,,	=	3·5 ,,
4 ,, ,,	=	4·4 ,,
5 ,, ,,	=	5·6 ,,

The method gives only a rough approximation : the ease with which cream rises depends on several varying factors, so that two samples of breast milk containing an equal quantity of fat may show a slight difference in the depth of the cream. As a simple clinical method, however, this may be useful for determining whether the milk is poor or rich in fat. Any milk which contains less than 2 cub. millim. of cream may certainly be considered deficient in fat.

The commonest and most serious defects in breast milk are excess of proteid and deficiency of fat. Upon one or other of these two faults depend many, perhaps most of the gastro-intestinal disorders of infants at the breast. It is a matter of the greatest practical importance in any case where an infant is not thriving at the breast to determine which constituent if any is at fault; too often weaning is recommended without any investigation of the milk, or without any attempt to rectify a fault which can sometimes be corrected by very simple means.

Excess of proteid is of course a relative term : the amount of proteid which can be digested varies with different infants. Excess may be suspected where the stool is pale, as if from undigested curd, and therewith the infant suffers from colicky pain, constipation, and perhaps vomiting of curd. Such a condition of milk may be habitual to a particular woman, and may make it almost impossible for her to nurse her infant ; more often, however, it is an induced condition, and probably depends in many cases on too frequent nursing. It has been found that the proteids in cow's milk are increased by increasing the frequency of milking, and from clinical experience it would seem that the same result occurs in human milk. So that excess of proteid may sometimes be reduced by lengthening the intervals between the feeds. In other cases an inactive life together with a diet too rich in proteid food is responsible for this condition of milk. Menstruation is sometimes the cause of a temporary increase in the percentage of proteids.

Any defect in the mother's habits or diet must be remedied, and if after this the infant is still unable to digest the proteid in the milk, the difficulty can sometimes be overcome by giving a tablespoonful of plain water or of barley water to the infant immediately before nursing; this seems to dilute the proteid, and in some cases entirely obviates the difficulty. If this fails, the administration of a small dose of sod. bicarb. (gr. ij.) with papain (gr. j.) immediately before feeding sometimes proves successful.

Deficiency of proteid seldom occurs apart from deficiency of fat, and when both are deficient the clinical results are generally due rather to the lack of fat than to the lack of proteid. Such a poor milk generally has a watery appearance, and is very apt to produce troublesome flatulence; the child may cease to gain weight, and may even waste although the flow of milk is abundant. In such cases the mother has sometimes been poorly fed, and the milk may be improved by increasing the proteids in the diet, and by attention to the mother's general health.

Excess of fat is quite uncommon and hardly ever produces any bad symptoms; but the writer has thought that occasionally troublesome vomiting may have been produced thus. A reduction of the quantity of food, especially of meat and eggs, taken by the mother may reduce the percentage of fat.

Deficiency of fat is much less rare, and may occur alone or in association with deficiency of proteid. Infants fed on such milk fail to increase in weight as they should, they suffer from chronic constipation and its attendant troubles, and may even show signs of rickets. It is often possible to detect any considerable deficiency of fat by a mere inspection of the milk, which has a watery, greyish appearance, but this of course gives only a very rough idea of the degree of deficiency. In any case where there is reason to suspect that the infant is suffering from such a deficiency of fat, an accurate analysis of the milk should be made if possible, or the fat may be estimated by the simple method suggested above. Human milk should certainly show not less than 3 per cent of fat, and any proportion lower than 2 per cent is likely to be associated

with some disorder or failure of nutrition in the infant. If it is found by repeated estimations that the proportion of fat is continuously low, an attempt should first be made to increase it by increasing the proteids in the mother's diet. For this purpose meat, poultry, or fish, and oatmeal porridge are valuable, and at the same time eggs and milk should be taken freely. As far as possible worry is to be avoided, and work or recreation must always stop short of fatigue. If these measures fail to increase the proportion of fat the deficiency must be made up by giving the infant two or three feeds daily of hot water and cream, or whey and cream (*see* "Artificial Feeding," p. 396), or if this is not convenient, cod-liver oil in doses of 10 to 20 drops according to the age of the infant may be given two or three times a day.

Deficiency of fat is not to be regarded as a reason for weaning: fat can readily be supplied in an easily digestible form by supplementing the mother's milk as suggested, whereas if the child is weaned entirely it becomes necessary to supply proteids also, and experience shows that one of the chief difficulties in artificial feeding is to supply the proteids in a digestible form.

Conditions in the Mother affecting the Milk.—The effect of menstruation and of pregnancy on lactation has already been considered. The milk may be diminished in quantity or lost altogether as the result of worry or mental shock. The quality of the milk is also sometimes seriously affected by any strong emotion, such as fright, anger, or grief, and under these conditions the milk may cause severe gastro-intestinal disturbance in the infant. Where this occurs the infant should be fed artificially for one or two days if necessary, and then again tried at the breast. In some very emotional women the milk fluctuates so frequently in quality that it may be necessary to wean altogether.

Effect of Drugs.—Certain drugs are found to be excreted partly in the milk, and may thus affect the infant. Of these the most important are belladonna and atropin, potassium iodide and bromide, salicylates, arsenic, and saline purgatives. There is some evidence that opium and morphia given to the mother may affect the infant, and in one case a fatal result has been recorded. Other drugs have occasionally been found in the milk, but not in sufficient amount to be of any practical importance.

The administration of drugs to the mother for the sake of their action on the infant has little if any advantage, and has very obvious disadvantages; besides the unpleasantness to the mother, the effect on the infant is very uncertain, and there is no means of accurately regulating the dose which the infant obtains; it is far simpler and more effectual to give the drugs to the infant in the usual way.

Weaning

Time of Weaning.—This must be determined chiefly from the circumstances of the individual case. As a rule an infant should not be weaned earlier than the end of the ninth month, nor later than the end of the twelfth month. It is very unadvisable to wean during the hot weather; in the writer's opinion it is often better to continue suckling even as late as the thirteenth or fourteenth month, if in this way weaning during the hot weather can be avoided. It is wise, also, not to wean at a time when teething is causing much disturbance; weaning should be done if possible in an interval between the cutting of teeth. Any transitory disorder, gastro-intestinal or otherwise, is usually sufficient reason for postponing the time of weaning. The conditions which may call for premature weaning have already been considered.

Method of Weaning.—Weaning may be sudden or gradual. The sudden cessation of suckling is hardly ever to be recommended, except in those cases where the obstinate refusal of the infant to take artificial food as long as it is suckled makes it necessary to stop breast-feeding altogether. Gradual weaning is more satisfactory in every way. The breast feeds are gradually replaced by artificial feeds, until at the end of four or five weeks all the meals consist of artificial food. The infant in this way gradually becomes accustomed to its altered diet, and digestion is much less likely to be upset. Many infants wean themselves: sometimes quite suddenly they seem to take a dislike to the breast, and refuse to suck; in some cases it is only one breast which the child refuses, and partial or entire suckling may be continued with the other. This refusal often occurs at a time when the milk is becoming scanty, at other times no cause whatever for the sudden dislike can be found.

Wet-Nursing

Where the mother is unable for any reason to suckle her infant the question arises whether a wet-nurse should be employed. There can be no doubt that, so far as the nature of the food goes, the only perfect way of replacing the mother's milk is by a wet-nurse; but the objections to this method are so very serious that it is now comparatively seldom used. The neglect of the nurse's own infant, possibly at the cost of its life; the immoral character of some of the women thus employed; the difficulty of excluding the presence of syphilis in women of whose previous history nothing is known;—these are some of the weightier objections, to which have to be added such minor points as the inconvenience to the household which the presence of such a nurse often involves, and the costliness of such a method of feeding.

If it is decided to employ a wet-nurse she must first be thoroughly examined by the

medical man, particularly for the presence of tubercle or syphilis. Quite recently the writer had under his care an infant whose wet-nurse had deliberately concealed the fact that she had hæmoptysis, and although she had been examined by a medical man, it was not until she had suckled the infant several months that the fraud was discovered by her becoming acutely ill with tuberculosis. The nurse's infant should also be examined if possible, partly to see whether it shows any evidence of syphilis, and partly to ascertain whether it has thriven on its mother's milk.

In the choice of a wet-nurse, her age and the age of her infant are of much less importance than the character of her milk, and this should be ascertained by the clinical methods described above, or better still by having an accurate analysis made. Inspection of the milk and also of the breasts, which should be full and firm, will also give some idea of the quantity and quality of the milk.

The diet of a wet-nurse must be regulated on the same lines as those laid down for the nursing mother, but with this caution, that as she has usually come from very poor circumstances, a too plentiful or too rich diet may easily alter the quality of her milk, so that it may disagree with her nursling. For the same reason, if she has been accustomed previously to hard, active life it is important that she should have plenty of exercise, otherwise the combination of a liberal diet with insufficient exercise is almost sure to produce milk too rich in proteid and fat.

The employment of a wet-nurse is by no means always a success; not every woman's milk agrees with every infant, and even if at first the nurse's milk agrees it is liable to any of the changes to which a mother's milk is subject, and may at any time cease to agree with the infant; and lastly, such women require constant supervision to see that they discharge their important duties faithfully.

Artificial Feeding

When the mother is unable to suckle her infant, the most readily available, and in most cases the best substitute for breast-feeding is cow's milk; but as will be seen, cow's milk requires modification to adapt it to the needs of the infant, and to carry out this modification intelligently some knowledge of the composition of cow's milk, and of the points wherein it differs from human milk, is essential.

Cow's milk, when first drawn from the udder, has an amphoteric reaction, but as it reaches the consumer it is generally faintly acid. Its specific gravity varies from 1029 to 1035. Microscopically it resembles human milk in consisting of a colourless plasma in which are suspended fat globules: the average size of these globules, however, is larger than in human milk. The plasma, as in human milk, contains proteids, sugar, and salts in solution, but the proportions are not the same as in human milk.

The composition of cow's milk varies considerably: the following average obtained from analyses made by several observers is therefore only approximate; the proportions in human milk are given here for comparison:—

Cow's Milk—	
Proteids—	
Casein, 3·25 / Lactalbumin, ·75	4·0 per cent
Fat	3·5 ,,
Sugar	4·0 ,,
Salts	·7 ,,
Water	87·8 ,,
Human Milk—	
Proteids—	
Casein, ·6 / Lactalbumin, 1·4	2·0 per cent
Fat	3·5 ,,
Sugar	7·0 ,,
Salts	·2 ,,
Water	87·3 ,,

It will be seen that the chief differences in composition are the much larger proportion of proteids and the much smaller proportion of sugar in cow's milk. But the proteids show an even more important difference, for while in human milk they consist chiefly of lactalbumin, in cow's milk they consist chiefly of casein: the proportion of casein to lactalbumin in cow's milk is about 4 : 1, in human milk it is about 1 : 2. The practical significance of this difference is readily appreciated if samples of the two kinds of milk are taken in two test-tubes, and some weak hydrochloric acid is added. Large solid masses of curd are at once formed in the cow's milk, while the human milk shows only very fine flocculent shreds of curd floating in it. Similar changes occur in the stomach, but the formation of curd here is due not only to the acid in the gastric juice, but also to a milk-curdling ferment, rennet. Another important difference is the sterility of human milk in contrast with the large number of bacteria present in cow's milk as it reaches the consumer; the alkalinity of human milk also contrasts with the acidity of cow's milk.

Modification of Cow's Milk.—It is obvious that in order to reduce the proportion of proteids to the standard of human milk some dilution will be necessary. For this purpose various diluents are available, but the choice of the diluent in any particular case is often a matter of considerable importance.

Plain water, as the simplest and least expensive, is most commonly used; such simple dilution not only reduces the percentage of proteid, but also materially reduces the size of the curd. In many cases this is all that is required to enable the infant to digest the casein; in others, however, this is not sufficient, and the digestion of the curd may be assisted by using some thickened fluid as a diluent.

Barley water is often used in this way, and whatever may be the rationale of its use, there can be no doubt that some infants will digest milk diluted thus when they are unable to digest milk diluted with plain water. It may be that such a thickened fluid does to some extent mechanically hinder the formation of large curds, but from a series of experiments made by the writer, it would seem that this attenuating action, if present at all, is extremely slight, and it has been denied altogether by some observers.

As a food in itself barley water has but little value; its most important constituent is starch, and although this is present in very small proportion, it sometimes causes trouble, especially in the earlier months of infancy. Flatulence and colic, and occasionally diarrhœa, have seemed in the writer's experience to be due to the use of barley water as a diluent. On the other hand, the laxative effect of barley water may be useful for an infant who is suffering from habitual constipation. Rice water and oatmeal water are sometimes used in the same way. The former has less laxative action than barley water, and may therefore be useful where there is a tendency to diarrhœa. Oatmeal water, although it has a distinct laxative effect, contains less starch than barley water, and may agree where barley water has failed. Gelatine is occasionally used in the form of a thin jelly, in the proportion of one teaspoonful to about four ounces of milk and water.

The methods of preparation of these diluents are as follows:—

Barley water is made by putting two teaspoonfuls of pearl barley into a pint of cold water, boiling slowly down to two-thirds of a pint, and then straining; or by mixing one teaspoonful of prepared barley (in powder) with one pint of boiling water, and then boiling for ten minutes. Whichever way it is made, a fresh supply must be prepared twice a day, and it must be kept in a cool place.

Rice water is made by soaking one tablespoonful of rice, which has previously been washed with cold water, in a quart of water at tepid heat for three hours; it is then boiled slowly for an hour, after which it is strained.

Oatmeal water is made by mixing one tablespoonful of oatmeal with a pint of boiling water, and boiling slowly for one hour; water must be added as necessary to make up the quantity after boiling to one pint; then strain.

Gelatine jelly is made by soaking one teaspoonful of gelatine in a teacupful of cold water for three hours, then standing it in hot water which is kept boiling until the gelatine is dissolved.

Another method which is occasionally useful, and which, perhaps, has some mechanical action as suggested above, is to add to the milk, after dilution with plain water, a very small quantity of some malted preparation of cereals, such as Mellin's food; half a teaspoonful or one teaspoonful of this added to the feed of milk and water will sometimes enable an infant to digest the casein of the milk.

Lime-water may often be used with advantage instead of, or in addition to plain water. This not only serves to dilute the proteids, but also seemed, in some experiments made by the writer, to have a definite though slight effect in diminishing the size and firmness of the curd; it also corrects the acidity of the cow's milk, and undoubtedly increases its digestibility. Lime-water should be used in the proportion of at least one tablespoonful to a three-ounce mixture of milk and water. A solution of sodium bicarbonate (gr. iv. to ℥j.) may be used similarly; it has the advantage that it does not cause constipation as lime-water often does. For this very reason, however, lime-water is usually to be preferred as a diluent in cases of diarrhœa, and in such cases two tablespoonfuls may be given in a three-ounce feed. The more concentrated form of lime-water known as liquor calcis saccharatus is sometimes more convenient; a teaspoonful of this to four tablespoonfuls of water gives a solution of nearly the same strength as the ordinary lime-water. A simpler way of using it is to add the saccharated lime solution directly to the food, for instance, to a three-ounce feed of milk and water fifteen drops of the liquor calcis saccharatus should be added.

The addition of an equal quantity of any of the diluents mentioned will reduce the proportion of proteids in cow's milk to 2 per cent, as in human milk; but the relative proportions of casein and lactalbumin remain exactly as before, so that the casein is still much more than in human milk; this can be seen from the comparison below. Experience shows that it is this excess of casein, and the large solid curd resulting therefrom, which is the chief difficulty in the use of cow's milk for infants' food, and in many cases an infant will not digest cow's milk until the dilution is carried so far that the *casein* is reduced to about the same percentage as in human milk,—in other words, the cow's milk has been diluted with three or even four times its quantity of the diluent.

Whatever dilution is used it is evident that all the other constituents of the milk are diluted at the same time, so that the percentage of fat which was previously the same as in human milk becomes too low, and the already insufficient proportion of sugar becomes even less sufficient. This may be seen from the following figures:—

Cow's Milk diluted with an equal quantity of Water—

Proteids—	
Casein, 1·63 } Lactalbumin, ·37 }	2·0 per cent
Fat	1·75 ,,
Sugar	2·0 ,,

Human Milk—		
Proteids—		
Casein, ·6 / Lactalbumin, 1·4	. . . .	2·0 per cent
Fat		3·5 ,,
Sugar		7·0 ,,

It is necessary, therefore, not merely to dilute the cow's milk in order to reduce the percentage of proteids, but also to add fat and sugar in order to bring the proportion of these up to the standard of human milk.

Addition of Fat.—The supply of a proper proportion of fat is a matter of the greatest importance in the feeding of infants. One of the commonest faults in infant feeding at the present time is the use of food containing too little fat, and many cases of chronic constipation, failure of nutrition, and rickets are due to this deficiency. Taking human milk as the standard, one would say that 3-4 per cent is the proper proportion, and although some infants will make satisfactory progress on less than this, it seems probable that 3 per cent is the minimum which should be allowed; certainly a proportion of fat below 2 per cent at any period during the first year of life is often associated with some disorder of nutrition.

If cream containing a known percentage of fat can be obtained it is a simple matter to correct the deficiency of fat in the milk. In towns where centrifugal machines are used in the separation of cream, it may be possible to obtain cream which contains a fairly constant average percentage of fat. Observations by the writer showed that fresh cream, as supplied by London dairies, contains on the average about 48 per cent of fat; but even such cream showed considerable variation (36·8-54 per cent) in its proportion of fat, and it is much to be desired that a standardised cream should be more generally obtainable. If the cream used contains 48 per cent fat, it is evident that one teaspoonful in a three-ounce feed will raise the fat 2 per cent; so that if used with a three-ounce mixture of equal parts of cow's milk and water, it will raise the proportion of fat from 1·75 to 3·75 per cent. Any desired percentage of fat can be obtained by similar calculation.

Cream is the upper portion of milk into which the fat globules have risen either (1) by gravity, that is, the milk has been allowed to stand until more or less of the fat has risen to the top, which has then been skimmed off as "cream," or (2) by centrifugalisation. The other constituents of milk remain practically unaltered, both in quality and quantity in the cream, and therefore, to be strictly accurate, it would be necessary to take into account the proteids and sugar of cream when we add it to the infant's food; for practical purposes, however, the quantity of the centrifugalised cream used is so small that in most cases the relatively minute proportion of those other constituents can be neglected, and only the fat in the cream needs to be considered.

Perhaps the simplest way to obtain a standardised cream is to prepare it at home by allowing a quart of milk to stand for three hours in a cool place, in a glass water-bottle such as can be obtained from any scientific instrument maker for three or four shillings, with a tap near the bottom of the vessel. After the milk has stood for three hours the lower portion is drawn off, and can be used as skimmed milk for domestic purposes, the upper part into which the fat has risen, the cream, is used for the infant. The percentages of fat thus obtained can be seen from the following table; they are only averages, for, as already mentioned, the rate at which fat rises depends on several varying factors; but these figures are sufficiently accurate for practical purposes, they were obtained with average London milk:—

Quantity of Upper Milk (Cream) from each quart of milk.	Proportion of Fat.
One pint	5·5 per cent
Fifteen ounces	6·5 ,,
Ten ounces	8 ,,
Five ounces	11 ,,

Such top-milk or cream can be used either as an addition to a mixture of milk and water to increase the fat, or more simply with direct dilution with water. Thus the addition of a tablespoonful of the 11 per cent cream to a three-ounce mixture of equal parts of milk and water, will raise the fat percentage from 1·75 to about 3·2; or the dilution of one part of this 11 per cent cream with two parts of water will give a fat percentage of about 3·7.

One great drawback to the use of mixtures which contain added cream is the difficulty, especially in towns, of obtaining it fresh. Amongst the poor also the expense makes it impossible in many cases to obtain cream.

A plain mixture of milk and water is therefore commonly used, and the results are sometimes quite satisfactory; but in a considerable proportion of cases the inevitable deficiency of fat in the mixture leads to some failure of nutrition, or to some degree of rickets.

Where no more accurate method can be carried out, it is well to adopt the simple plan of allowing the milk to stand in a jug for an hour or more, and then, with as little shaking as possible, pour off the top half to be used in preparing the infant's food; a valuable increase of fat is obtained in this way.

Where even this is impossible the milk should be given with the least possible dilution consistent with the digestion of the casein, and, if necessary, the deficiency of fat must be made good by the daily administration of small doses of cod-liver oil. The writer has sometimes used a little butter, a piece about the size of a large pea, dissolved and shaken up well, in a three- or four-ounce feed of diluted milk; the propor-

tion of fat may be raised in this way nearly 1 per cent. Infants generally tolerate this amount of butter well, but the method is not to be recommended where cream can be obtained, for the butter does not mix easily with the milk, it has a taste which may be disliked, and it is often adulterated, especially with preservatives which may do harm.

Addition of Sugar.—Whatever strength of cow's milk is used the addition of sugar is necessary to bring the proportion up to that found in human milk. For this purpose milk-sugar is preferable to cane-sugar. A standardised solution of milk-sugar is sometimes used for the dilution of milk, but this has the disadvantage that the solution must be frequently made afresh to prevent its undergoing fermentation. It is quite as simple and usually much more convenient to add the milk-sugar directly to the diluted milk; and if it be remembered that one level teaspoonful (75 grains) of the powdered milk-sugar in a three-ounce mixture gives a proportion of about 5 per cent of sugar the calculation is simple enough. Thus in a three-ounce mixture of equal parts of milk and water, which, as already pointed out, contains 2 per cent of sugar, the addition of one level teaspoonful of milk-sugar will raise the proportion to 7 per cent; and in 6 oz. of such a mixture the addition of two teaspoonfuls of milk-sugar will have the same effect.

The use of milk-sugar is not a matter of great importance, cane-sugar will serve the purpose almost equally well, and amongst the poorer classes it is commonly used on account of its cheapness. If ordinary white lump sugar is used a lump about half an inch square should be used to a three-ounce mixture of milk and water.

Milk Mixtures.—Of recent years there is a growing tendency to the use of carefully calculated formulæ in the modification of milk for infant feeding, with the object of approximating more nearly to the standard of human milk. This, undoubtedly, is a step in the right direction, and it is much to be desired that such careful modification should be more generally carried out; but at the same time it must be remembered that an infant is not a test-tube, and that no amount of mathematical precision in the calculation of formulæ will eliminate the idiosyncrasies of the infant. We may determine to a nicety the percentage of each constituent in a mixture, but the much more serious difficulty still remains to know what particular percentage will suit any given case. There is no golden rule whereby to determine this; one can judge to some extent from the age of the infant, but the requirements of each individual case must remain to a large extent a matter of experiment. The proportions of fat and sugar which are required vary very little at any period during the first year of life; the former should be 3-4 per cent, the lower proportion being given during the first few weeks, the sugar should be 6-7 per cent. The proportion of proteid is the chief difficulty; the following proportions can only be taken as a rough guide, for infants of the same age differ considerably in their power of digesting the proteids of cow's milk:—

Age.	Percentage of Proteid.
1st week	1 or less
2nd week to 6th week	1·5
6th week to 3rd month	1·75
3rd month to 6th month	2·0
6th " 8th "	2·5
8th " 12th "	3·0

These proportions can be approximately obtained by diluting the milk according to the following table:—

	Milk.	Water.
1st week	1	3
2nd week to 6th week	1	2
6th week to 3rd month	2	3
3rd month to 6th month	1	1
6th " 8th "	2	1
8th " 12th "	3	1

If the milk is diluted in this way, and the quantity of each feed is regulated according to the table given below, the addition of one teaspoonful of 48 per cent centrifugalised cream, and of milk-sugar in the proportion of about one teaspoonful to every 3 oz. of the mixture, will give satisfactory proportions. For example:—

Formula.	Approximate Proportion. Per cent.
1st week—	
Milk, 1 tablespoonful	Proteid, 1
Water, 2 tablespoonfuls	Fat, 3·8
Lime-water, 1 tablespoonful	Sugar, 6·5
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, half a teaspoonful	
(Half this quantity to be given as a feed.)	
2nd to 6th week—	
Milk, 2 tablespoonfuls	Proteid, 1·3
Water, 3 "	Fat, 3·2
Lime-water, 1 tablespoonful	Sugar, 6·3
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, 1 level "	
6th week to 3rd month—	
Milk, 3 tablespoonfuls	Proteid, 1·6
Water, 3½ "	Fat, 3·2
Lime-water, 1 tablespoonful	Sugar, 6·5
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, 1 level "	
3rd to 6th month—	
Milk, 4 tablespoonfuls	Proteid, 2·0
Water, 3 "	Fat, 3·25
Lime-water, 1 tablespoonful	Sugar, 6·5
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, 1 level "	
6th to 8th month—	
Milk, 8 tablespoonfuls	Proteid, 2·6
Water, 2 "	Fat, 3·4
Lime-water, 3 tablespoonfuls	Sugar, 7·0
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, 1½ "	
8th to 12th month—	
Milk, 12 tablespoonfuls	Proteid, 3
Water, 2 "	Fat, 3·4
Lime-water, 2 tablespoonfuls	Sugar, 6·2
Cream (48 p.c.), 1 teaspoonful	
Milk-sugar, 2 "	

If a weaker cream is used, so that larger quantities are required, it becomes necessary to take into account the proteid and sugar as well as the fat in the cream; and often it is convenient to use such a thin cream instead of milk as the basis of the mixture. The proteids and sugar in any cream are practically in the same proportion as in milk. An 8 per cent cream can be obtained as described above by taking the upper 10 ounces from a quart of milk which has been allowed to stand for three hours. Such a cream only requires dilution and the addition of sugar. For an infant between the ages of 6 weeks and 3 months such a mixture as the following is suitable:—

Formula.	Approximate Proportion. Per cent.
Cream (8 p.c.), 3 tablespoonfuls	Proteid, 1·7
Water, 3 tablespoonfuls . .	Fat, 3·3
Lime-water, 1 tablespoonful .	Sugar, 6·6
Milk-sugar, 1 teaspoonful	

At six months it may be used with an equal quantity of diluent, thus:—

Formula.	Approximate Proportion. Per cent.
Cream (8 p.c.), 6 tablespoonfuls	Proteid, 2
Water, 5 tablespoonfuls . .	Fat, 4
Lime-water, 1 tablespoonful .	Sugar, 7
Milk-sugar, 2 teaspoonfuls	

Similarly by using the upper 15 ounces (6·5 per cent fat) from the quart of milk, and diluting with an equal quantity of water, a mixture is obtained containing proteids 2 per cent, fat 3·25 per cent; or, by using the upper 5 ounces (11 per cent fat), and diluting with twice the quantity of water, a mixture is obtained with proteids 1·3 per cent, fat 3·7 per cent.

The percentages obtained by any such methods, even with the utmost care, are necessarily only approximate, for the composition of milk, even from the same cow, varies not merely from day to day, but even at different hours of the day; and although this variation is somewhat less in the mixed milk of commerce, it is still present, and in cream it is still more marked.

Some of the large London dairy companies now undertake the preparation of milk mixtures according to prescriptions from medical men, and this at once simplifies the matter; but the costliness of such a luxury places it at present beyond the reach of any but the wealthier classes. Home modification is much less expensive, and is quite satisfactory where proper care is taken. For the hard-working poor even such modification may be impossible, and for them any elaborate formulæ are mere counsels of perfection. Simple dilution with plain water, or barley water, according to the table given above, with the addition of a small lump of white sugar to each feed, often gives very good results; and if the deficiency of fat be made good by daily administration of cod-liver oil, infants fed thus may do perfectly well.

Quantity and Frequency of Meals.—Whatever food is used it is most important that feeding should be regular. The tendency is to feed an infant too often and with too large quantities. The infant who is fed "whenever it cries" cannot be expected to digest its food, and it is not surprising if an infant vomits when it is allowed to take a much larger quantity than the stomach can possibly hold without considerable over-distension. The average capacity of the stomach, according to Dr. Holt, is at birth $1\frac{1}{5}$ oz., at three months $4\frac{1}{2}$ oz., at six months 6 oz., and at twelve months 9 oz. Some post-mortem observations made by the writer agreed fairly closely with these measurements, and clinical experience has shown that at these ages these are about the quantities which a healthy infant is able to digest. Of course some latitude must be allowed for the constitution of the particular infant: a vigorous baby above the average size may be able to digest and may require a larger feed than a smaller infant of the same age. Some infants are habitually "small feeders," and will thrive on smaller quantities than those mentioned.

The following table may serve as a guide for the feeding of an average healthy infant:—

Age.	Interval between Feeds.	Quantity.	Number of Meals.	Total Amount in 24 hours.
First week	2 hours	1 oz.	10	10 oz.
2nd week to 6th week . .	2 ,,	$1\frac{1}{2}$ to 2 oz.	8	12 to 16 oz.
6th week to 3 months . .	$2\frac{1}{2}$,,	3 to 4 oz.	6	18 to 24 oz.
3 months to 6 months . .	3 ,,	4 to 6 oz.	6	24 to 36 oz.
6 months to 8 months . .	3 ,,	6 to 7 oz.	6	36 to 42 oz.
8 months to 12 months .	3 ,,	7 to 8 oz.	5	35 to 40 oz. (with additional food)

The infant should be accustomed, as in breast-feeding, to sleep several hours at night without being fed; the interval should be increased gradually from four hours during the first week up to six hours at three months, and with some infants seven or eight hours at six months.

Boiling, Pasteurisation, and Sterilisation of Milk.—Cow's milk and cream as they reach the consumer contain a considerable number of bacteria, and it has been proved beyond dispute that some of the acute specific fevers, particularly typhoid, scarlet, and diphtheria, have

occasionally been conveyed by milk; recently, also, much attention has been paid to the transmission of tuberculosis in this way, and it has been shown that milk from cows with tuberculosis of the udder frequently contains the tubercle bacillus; the risk of infection is undoubtedly a real one, but the frequency of its occurrence has probably been greatly exaggerated. A much commoner and more widely disastrous infection from milk is that which produces summer diarrhœa; whatever may be the micro-organism which produces it, there can be little doubt that milk infection is chiefly responsible for the terrible mortality from infantile diarrhœa. (*See* "Gastro-intestinal Disorders of Infancy.")

For the prevention of these various risks three methods of preparing the milk are available: (1) Boiling, (2) Pasteurisation, (3) Sterilisation.

Boiling is at once the simplest and the least objectionable method of preparing the milk; but it must be understood that by "boiling" the writer means simply heating the milk just to the boiling-point, and then at once stopping the process. It is advisable to stir the milk frequently during the heating, so that the whole of it may be heated to the boiling-point. Bacteriological observations have shown that for all practical purposes milk which has been treated thus is a perfectly safe food so far as any risk of infection is concerned; even virulent tuberculous milk was found to be innocuous after exposure to a temperature of only 185° F. for five minutes (Woodhead). Prolonged boiling for five, ten, or fifteen minutes, and *a fortiori* for such periods as half an hour or one hour, is not only unnecessary, but in some way seems to impair the nutritive value of the milk. A sufficient quantity of the food to last twelve hours can be prepared twice daily; the milk and cream should be mixed before the boiling is carried out, but if lime-water is to be used it should be added subsequently, as some chemical change seems to be produced by boiling it with the milk.

After boiling, the mixture should be put at once into clean bottles, which are closed with tight-fitting stoppers, and should be rapidly cooled in cold water, or on ice, and then kept in some cool place until required.

Compared with pasteurisation boiling has the disadvantage that it gives the milk a taste which some infants may dislike, but, as a matter of fact, very few infants object to it, so that this is seldom a practical difficulty. The coagulation of the lactalbumin by boiling is also a disadvantage, as it reduces to some extent the nutritive value of the milk.

Prolonged boiling, like other methods of complete sterilisation, has been shown to produce some change in the milk whereby it loses its antiscorbutic power; and the continuous use of such milk is liable to produce infantile scurvy. So far all are agreed; but there is more to be said on this point. Infants come under notice every now and then who have been fed continuously on milk which has been boiled for fifteen minutes, or perhaps longer; they show no evidence whatever of pronounced scurvy, but they are "not getting on," they are peevish and miserable, their skin, instead of showing a clear healthy colour, shows an earthy pallor, only less marked than the anæmia of scurvy, their muscles are flabby, and they are not gaining weight as they should; there is, in fact, a cachexia, which is hardly defined enough to be called scurvy, but may well be the vanishing point of scurvy—and this appears to be due in some cases to overboiled milk, but much more often to sterilisation. If, however, milk be heated only just to the boiling-point, and then immediately cooled, it would seem that the risk of any such cachexia is considerably diminished.

Pasteurisation of milk consists in keeping it at a temperature of 155°-165° F. for twenty to thirty minutes. This is found to render the milk innocuous, so far as pathogenic micro-organisms are concerned, but like heating just to the boiling-point, or even boiling for two or three minutes, it does not necessarily render the milk "sterile" in the strict sense of the word.

Various forms of apparatus are made for this purpose under the name of "sterilisers," this term being loosely used to include both pasteurising and sterilising apparatus. The principle is the same in most of these forms: the milk is placed in an inner vessel which is surrounded by a jacket of cold water, into which a thermometer passes, the water is slowly raised to a temperature of 155°-165° F., and the source of heat is then removed; the milk is left surrounded by this heated water for twenty minutes. An ordinary deep tin saucepan, with a wire or perforated tin tray suspended in it, as in a fish-kettle, so that the bottle of milk may not be overheated by contact with the bottom of the saucepan, will suit the purpose admirably; a hole is bored in the lid, and a high temperature thermometer, which can be bought from any scientific instrument maker, passes through a cork fitted into this hole, into the water inside the saucepan. A glass bottle which contains the milk, and is stoppered with a plug of cotton wool, is placed on the tray in the saucepan, and surrounded with cold water up to the level of the milk; the saucepan is then placed on the fire, or more conveniently over a gas-jet, and the contents are slowly heated until the thermometer projecting through the lid registers 160° F., the saucepan is then removed from the fire, and after twenty minutes the bottle of milk is taken out, the cotton wool is replaced by a tight-fitting stopper, and the bottle is then rapidly cooled, and kept in some cool place until required for use.

Whether pasteurisation is altogether free

from the risk of producing the cachectic condition which may result from prolonged feeding with sterilised milk, and, to a less degree, from the use of overboiled milk, is at least open to doubt.

Sterilisation means, properly speaking, the destruction of all bacteria and spores, so that milk thus treated will keep indefinitely. This is accomplished by subjecting the milk to prolonged boiling in the usual way for an hour or more, or by the use of superheated steam. For domestic purposes an apparatus exactly similar to that used for pasteurising is most convenient; the temperature, however, is raised to 212° for not less than forty minutes. Although in most cases this will render the milk completely sterile, it does not necessarily do so, the spores of some micro-organisms resist such a temperature for a much longer time.

Such a method of preservation is now widely practised for commercial purposes; sterilised milk can be obtained in most towns, and it is to be remembered that "humanised milk," as sold in the shops, is also sterilised. These sterilised preparations are very useful where, for any reason, for example in travelling, it is impossible to obtain fresh milk. In some cases, also, sterilised milk seems to be more easily digested than milk prepared in other ways, and some observers have obtained good results by feeding infants on undiluted milk which has been heated to 212° F. for forty minutes.

But what has been said of overboiled milk applies with much greater force to milk sterilised by prolonged exposure to a high temperature. The writer's experience has convinced him that the prolonged use of sterilised milk is responsible, not only for that comparatively rare condition which we recognise as fully developed scurvy, but also for the less rare but not less important condition of malnutrition and cachexia which has been described above. In the present state of our knowledge it is impossible to explain why only a limited number of the cases fed thus develop these untoward symptoms, but it is very important that their relation to such feeding should be recognised.

To sum up, one may say that either heating just to the boiling-point, or pasteurisation, is much to be preferred to sterilisation, using this term in its strict sense. All these methods of preparation are to be regarded as necessary evils, not as virtues in themselves. The ideal to be aimed at is not the destruction of micro-organisms by processes which may, and almost certainly do, diminish the nutritive value of the milk, but the prevention of such contamination by properly regulated sanitation in cow-sheds and in the purveying of milk. Lastly, it may be pointed out that no method of preparation can make a bad milk good; a milk which is "only slightly turned" remains unfit for infant food whatever may be done to it, and a milk which is poor in fat does not become any the richer by being "sterilised."

Subsequent Care of the Milk.—Whatever precautions are taken in preparing the milk, it must be remembered that if exposed to the air afterwards it quickly becomes contaminated again with micro-organisms, and decomposes quite as rapidly, if not more rapidly than fresh milk. The jug in which it is placed should be cleansed with boiling water just before and after use, and, so long as the milk is in it, should be kept covered in a cool place. A better way is to use bottles properly cleansed with boiling water, which can be stoppered with sterilised wool, or with some tight-fitting stopper. In many of the special forms of apparatus now sold for pasteurising or sterilising, the milk to be prepared is already placed in feeding-bottles stoppered with wool or special stoppers, so that it is only necessary to replace the stopper by a teat when the infant is to be fed.

Feeding-Bottles and their Use.—The best form of bottle is the boat-shaped feeder, to one end of which a teat is directly attached. A rubber tube is to be avoided, it is almost impossible to clean it thoroughly, it becomes coated with stale milk and bacteria, and is a fruitful source of danger. Before and after use the bottle should always be well washed with boiling water, and in the intervals between feeds both bottle and teat should be kept in water to which a good pinch of borax has been added. The teat especially must be carefully cleansed, the inside is apt to become foul. Recently a mere conical cap which can be turned inside out for cleansing purposes has come into use, but it has the practical disadvantage that it lacks a shield, and a vigorous baby will sometimes nearly choke itself by cramming the whole of the teat into its mouth.

The size of the holes in the teat is a matter of some importance. Vomiting is sometimes due to the use of a teat in which the holes are so large that the child gets its food too quickly. On the other hand, the writer was recently consulted by an anxious mother on account of the supposed sudden failure of appetite in her infant. After sucking at the bottle a few minutes it would cry fretfully and refuse it. Examination of the teat, which was a new one, showed that the holes were so small that it was only just possible by very forcible sucking to get any milk through. Enlargement of these with a pin speedily set matters right. It makes little difference whether the holes are simple round punctures or of leech-bite shape so long as the child can draw the milk at a suitable rate.

The milk should be warmed to about 100° F. just before the meal; this may be done by standing the bottle, up to the level of the surface of the milk, in a jar of water as hot as the hand can comfortably bear for about five

minutes, or more accurately by such an apparatus as has been described above for pasteurising, the thermometer being kept at 100° F. for about three minutes.

The bottle must be gradually tilted by the nurse as the child empties it, so that the nipple end shall be kept full to prevent the child sucking in air. The infant should be accustomed to take its food without interruption, the whole meal lasting ten to twenty minutes, after which any remaining milk must be thrown away, and the bottle washed and put away. The infant should not be allowed to take its food in a desultory way, lingering over its bottle for half an hour or more, or still worse, being allowed to suck even after the bottle is empty in order to keep it quiet.

Additional Food.—Supposing an infant to be making satisfactory progress on diluted cow's milk, the question arises, How soon should any additional food be given? As a general rule no other food of any kind is required until the infant is at least seven months old, and many infants will do well without any addition to their diet until nine months old. The best guide is the weight of the infant, so long as this is increasing satisfactorily the diet is sufficient.

As early as the third or fourth month a small quantity of a completely malted food, such as Mellin's, can be added to one or two of the feeds daily without doing any harm, but it is seldom necessary. At the age of six or seven months one of the partially malted foods, such as Allenbury No. 3, or Nestlé's Food, which contain some unconverted starch, may be cautiously added to one or two meals in the day. If these are well taken, some prepared wheat flour or a Robb's biscuit may be given once or twice a day at the age of nine or ten months, and about the same time the yolk of an egg lightly boiled may be given occasionally. At the age of twelve months a tablespoonful of crumbled stale bread or a rusk soaked in milk or in red gravy from underdone meat may be allowed once a day, and a little later mealy potato mashed into a fine flour and beaten up with milk may be given two or three times a week.

Milk of Animals other than the Cow

Asses' Milk, Goat's Milk.—Unfortunately there are some infants who will not thrive on cow's milk however carefully it is diluted, and for such it is necessary to find some other method of feeding.

For those who are able to afford it the milk of the ass or the goat is sometimes successful as a substitute for mother's milk, but by no means always, and such milk should never be ordered without due regard to the cause of the failure to thrive on cow's milk. This will be better understood from a comparison of the analyses of these various milks:—

	Human Milk.	Cow's Milk.	Asses' Milk (Winter-Blyth).	Goat's Milk (Winter-Blyth).
Proteid	2·0 {Casein, ·6; Lact-albumin, 1·4}	4·0 {Casein, 3·25; Lact-albumin, ·75}	1·8 {Casein, 1·0; Lact-albumin, ·8}	3·7 {Casein, 3·0; Lact-albumin, ·7}
Fat	3·5	3·5	1·0	4·2
Sugar	7·0	4·0	5·5	4·0
Salts	·2	·7	·4	·5

(Under the head of lactalbumin in the analyses of asses' milk and goat's milk peptones amounting to about ·1 per cent have been included for convenience.)

It will be seen that asses' milk is much poorer than cow's milk both in proteids and in fat (in a specimen examined by the writer the fat amounted to only ·3 per cent). The casein forms a very fine flocculent curd almost like that in human milk, and therefore may be digested well, where, as usually happens, the trouble in digesting cow's milk is due to the casein. Its extreme poorness in fat, however, makes it unsuitable for continued use, and asses' milk must be regarded only as a temporary food suitable for very young or very weakly infants, or for such a feeble condition of digestion as is left after a severe attack of gastro-enteritis. It has a laxative action, which is also sometimes useful, but may make it unsuitable where there is any tendency to diarrhœa.

Goat's milk, on the contrary, is almost as rich in casein as cow's milk and is richer in cream. It might be expected, therefore, that it would be useless ordering goat's milk where there is difficulty in digesting casein; this, however, does not necessarily follow, for in some observations made by the writer, the curd given by goat's milk with hydrochloric acid was much more finely divided than that given by cow's milk under similar conditions. A trial of goat's milk may therefore be useful even where the infant is unable to digest the casein of cow's milk. Some infants thrive excellently on goat's milk, and in the country where a field is available it is not an expensive matter to keep a goat. One infant under the writer's care was fed on goat's milk for several months; the goat cost fourteen pence a week to keep, and yielded at least two to three pints of milk twice a day. Goat's milk, of course, requires dilution in the same way as cow's milk.

Ewe's Milk and Mare's Milk have also been used for infants. The former is even richer in fat and casein than goat's milk, and is hardly likely to suit an infant who cannot digest cow's milk. Mare's milk is slightly poorer in fat and casein than cow's milk, and occupies in this respect an intermediate position between cow's milk and asses' milk.

With the more intelligent modification of cow's milk which is practised nowadays, the necessity for the use of the milk of other animals has become less, and it is now comparatively seldom that any of those mentioned here are used.

Milk Preparations

"*Humanised Milk*," as sold by many of the larger dairy companies, is of considerable value as a temporary food, where for any reason it is impossible to carry out the careful modification of cow's milk at home.

The term "humanised" simply means that the percentages of fat, proteid, and sugar have been modified to bring the milk as near as possible in composition to human milk, and the principles of preparation are exactly the same as those already described for the home modification of milk. Some humanised milk, however, has its digestibility further increased by partial pancreatisation, a process which will be described below.

Humanised milk is now commonly sold in two or three different strengths, so that the percentage of proteid can be increased as the infant's powers of digestion increase.

As a temporary measure such milk is often of great value, and the writer has obtained excellent results from it; but it is apt to be forgotten that these commercial preparations have been sterilised, usually by prolonged exposure to high temperature, and have not therefore the full value of fresh milk. In this way they are inferior to the home modifications of fresh milk. It happens not uncommonly that an infant thrives well for a few weeks, or two or three months, on "humanised milk," and then begins to fail and to show the same vague symptoms which have already been described as due to the prolonged boiling or sterilising of milk. These sterilised preparations, therefore, should not be used alone for long periods; it is preferable, if possible, to modify fresh milk at home as directed above.

Peptonised Milk.—Under the name of peptonisation are generally included two different processes for the predigestion of foods. In the one the ferment of the gastric juice, pepsin, is used; in the other the ferments of the pancreas. The latter is by far the more convenient to use for the predigestion of milk, for not only is it said to act with special ease on the casein of cow's milk, but it also acts in an alkaline or neutral medium, whereas pepsin requires a distinctly acid medium. By this predigestion the proteids including the casein are more or less completely converted into soluble peptones which are easily absorbed.

Various methods of peptonisation are in vogue. A very simple and effectual method is to mix a quarter of a pint of boiling water with half a pint of cold milk, then add the following powder: Extract. pancreatis (Armour's) gr. ijs., sod. bicarb. gr. x., and allow to stand in front of the fire for ten minutes with occasional stirring, then heat the mixture just to the boiling-point. This final heating stops the action of the ferment, which if continued too long gives an unpleasant taste to the milk; the heat at the same time serves to destroy any micro-organisms in the milk. Fairchild's peptogenic milk powder is very similar in composition, and used according to the directions supplied with it produces an easily assimilated milk mixture, in which the casein is partially digested, and which, according to Dr. Leeds's analysis, closely resembles human milk in its composition. Benger's liquor pancreaticus is used thus: Half a pint of boiling water is mixed with a pint of cold milk, two teaspoonfuls of the liquor with 20 grains of sodium bicarbonate are added, and the whole mixture is kept in front of a fire for about one hour and then boiled. If used without further dilution this mixture requires the addition of about half a teaspoonful of milk-sugar to every three ounces. Fairchild's zymine (gr. v. with sod. bicarb. gr. xv., the contents of one of the glass tubes in which it is sold) can be used similarly.

Whatever method is adopted the degree of peptonisation can be regulated by the time the peptonising process is continued. In discontinuing the use of peptonised milk it is often useful to reduce the time of peptonisation gradually, so that more and more of the work of digestion may be left to the infant's stomach. In this way many an infant who seems unable to digest cow's milk may gradually be coaxed into digesting it without artificial aid.

For some conditions peptonised milk is particularly valuable. The miserable emaciated infant, who, in spite of much care in the dilution of milk, is continually whining or screaming with flatulence and colic, whose bowels are costive, and stools consist largely of undigested curd, will often turn the corner and make rapid progress on peptonised milk; so, too, the infant who is just convalescent from an attack of gastro-enteritis, and whose stomach, as so often happens, seems to have lost in great measure its digestive power, is often benefited by the use of peptonised milk for two or three weeks.

But there are certain disadvantages attaching to peptonised milk which it is well to remember. It has a laxative effect which is not always desirable, and in some infants causes troublesome diarrhœa. It must, therefore, be used

with caution where there is any tendency to looseness of the bowels. If this predigested milk is used too long it is sometimes very difficult to return to ordinary milk, the stomach refuses to do its work after having it done for it so long. Lastly, in the process of peptonisation the milk loses its antiscorbutic value, and an infant fed for several weeks on peptonised milk may develop infantile scurvy.

Whey is often very useful for infants who are unable to digest casein. It is made either by the addition of an acid or by the action of rennet on milk. A convenient method is to use essence of rennet or Benger's curdling fluid. Add one teaspoonful of either of these to half a pint of milk, which has been warmed to about 98° F.; let it stand before the fire until the curd has set, then break up the curd thoroughly with a fork, and allow it to stand for fifteen minutes, then strain off the curd, and heat the whey to the boiling-point.

Where rennet or essence of rennet is not obtainable, lemon juice may be used in the proportion of one teaspoonful to half a pint of milk; such lemon whey is particularly useful as a food in infantile scurvy. In some cases sherry whey is more useful, especially where it is desired to combine a stimulant with the food; in the extreme exhaustion of acute gastro-enteritis, or in the late stages of marasmus, few foods are so well taken. For infants, also, whose digestion has become so enfeebled by a long course of unsuitable diet that they seem scarcely able to digest anything, the sherry whey seems to act both as a stimulant to the gastric function and an easily assimilated food, so that, after a few days, it is possible to add cautiously other food, and so gradually to encourage the infant to take a more nourishing diet. Sherry whey is made by heating half a pint of milk just to the boiling-point, adding a good wineglassful of sherry, then heating to the boiling-point and allowing to stand until the curd has settled; the curd is then separated from the whey by straining through muslin.

Whey is milk from which the casein has been removed; but the casein in its precipitation carries with it the greater part of the fat globules, so that the constituents of whey are the lactalbumin, sugar, and salts of milk, with very little of the fat and practically none of the casein. It is, therefore, not sufficiently nourishing for prolonged use; the proportion of fat, in particular, is seriously deficient; it amounts, according to Koenig, only to ·24 per cent, while the proteid amounts only to ·82 per cent. If whey is to be used for more than a few days, therefore, it is generally advisable to add cream; and many infants will make excellent progress for several weeks on a mixture of whey and cream alone. The addition of a little raw meat juice to two or three feeds, or the substitution of one or two feeds of veal broth or chicken broth, will rectify the low percentage of proteids. A useful mixture may be obtained by adding cream and sugar in the following proportions:—

Whey	4 tablespoonfuls.
Cream (48 per cent) . .	1 teaspoonful.
Milk-sugar	Half a teaspoonful.

For an infant who is able to digest more proteid a small quantity of casein may be added by using the weaker home-made 8 per cent cream; thus—

Whey	6 tablespoonfuls.
Cream (8 per cent) . .	2 ,,
Milk-sugar	1 teaspoonful.

When the digestion is still further improved, ordinary milk may be added to the whey (after this has been boiled to stop the action of the curdling ferment), and such a mixture as the following may be given:—

Milk	3 tablespoonfuls.
Whey	3 ,,
Cream (48 per cent) . .	1 teaspoonful.
Milk-sugar	1 ,,

Where cream or milk is used with whey it is necessary to use rennet-made whey which has been boiled—not the acid or sherry whey, for in these the acidity may curdle the added cream or milk.

Koumiss may be mentioned here, but is rather suitable for children over the age of two years than for infants. As originally used by the Tartars, it was made from mare's milk or asses' milk, with kefir grains, consisting of yeast and bacteria. In this country it is made from cow's milk with ordinary brewers' yeast.

At the Hospital for Sick Children, Great Ormond Street, a not unpleasant preparation has been made by the following method:—Mix together new milk 40 oz., water 40 oz., brewers' yeast 1 oz., and loaf sugar 3 oz., in an open jar; allow the mixture to stand at 30° C. for five or six hours. When bubbles of gas begin to rise in it, add milk 40 oz., water 40 oz., and milk-sugar $4\frac{1}{2}$ oz.; then bottle, being careful to secure the corks with string or wire. Keep in a cool place for five days before using. It must be tapped with a champagne cannula, not by removing the cork. This has been used with satisfactory results for older children in convalescence from acute disease, and is sometimes useful for infants with troublesome vomiting. It contains alcohol in very small quantity, 1-2 per cent, about a quarter of the amount present in ordinary London porter, and therefore is to some extent a stimulant. It has a somewhat beery taste, and is markedly acid. It is very deficient in fat, and in this respect is a poor food for young children, except for very short periods. Prepared according to the method described above, it contained, in two samples examined by the writer, 1 per cent of fat, and about the same proportion was found in samples obtained from shops; but one supplied by a

London dairy company contained only ·05 per cent of fat.

In these days of sterilisation, it is interesting to note that the value of koumiss as a food in certain cases is undeniable; but none the less bacteriological examination showed in all the specimens examined by the writer that it was swarming with micrococci, as appears to be always the case from its method of preparation.

Condensed Milk.—This is cow's milk which has been evaporated *in vacuo*, and then sterilised by heat. It is sold in two varieties—(1) sweetened, (2) unsweetened. The better-class brands are made from whole milk, *i.e.* milk from which none of the cream has been removed, or even from milk to which cream has been added; but a very large quantity of the cheap condensed milk is made from skimmed or separated milk which has lost nearly all its cream, and which, therefore, even if it were fresh, would be totally unfit for infant food.

The ordinary sweetened condensed milk contains a large quantity of added cane-sugar. The average composition of the better-class brands, as calculated from published analyses quoted by Blyth, is as follows:—

Proteids	10·9 per cent
Fat	9·5 ,,
Sugar— Cane-sugar, 41·1; Milk-sugar, 13·1	54·2 ,,
Salts	2·03 ,,
Water	21·37 ,,

It will be seen that in order to reduce the percentage of sugar to the correct proportion, about 7 per cent, it is necessary to dilute sweetened condensed milk with seven times its quantity of water, with the following approximate result:—

Proteids	1·3 per cent
Fat	1·1 ,,
Sugar	6·7 ,,
Salts	·2 ,,
Water	90·7 ,,

Such a mixture contains altogether too little fat, but the directions on some of the brands recommend even further dilution. For instance, in one (Sledge Brand) examined by the writer, the fat amounted to 9·5 per cent, the directions recommended dilution with 11 parts of water for the first month, and then with 8 parts of water for the second and third months, making the proportions of fat respectively ·8 per cent and ·1 per cent. The proteids, also, with such dilution, become very deficient. As a matter of fact these directions are rarely carried out with any accuracy, for the milk is measured with a teaspoon, which is then used to stir it into the water; condensed milk, being very thick, clings to the teaspoon, which may thus contain as much as 3 or 4 drachms. In this way the writer has found the percentage of fat, where condensed milk was prepared nominally in the proportion of a teaspoonful to 4 ounces of water, to be as much as 3·2 per cent. In such a case, however, the lessened dilution of fat meant also lessened dilution of sugar, which was altogether in excess (probably about 18 per cent). There is, in fact, no *via media* in using the ordinary sweetened condensed milk, either the sugar must be seriously in excess, or the fat must be seriously deficient.

With the inferior sweetened condensed milk which the poor buy at about half the price of the better-class brands, the deficiency of fat is even worse. The proportion of fat in some of these was as low as 0·4 per cent (Dyer), and as the sugar is as excessive as in the better-class brands they require similar dilution; the result is a proportion of fat, in some of these, amounting to barely 0·5 per cent. It is hardly to be wondered at that rickets and marasmus should result from such a diet.

Unsweetened condensed milk of a reliable brand is much more satisfactory in this respect; it contains no cane-sugar, and requires much less dilution, so that the percentage of fat is not so greatly diminished. In the manufacture of some of these brands the fat has been increased by the addition of cream before condensation. The Viking Brand, in the sample examined by the writer, contained 11 per cent of fat; the "Ideal" contained 10·4 per cent; the "First Swiss" (Romanshorn), 9·4 per cent. These require dilution with about three times their quantity of water to bring the other constituents to suitable proportions: the percentage of fat thus becomes 2·75, 2·6, and 2·35 respectively. Milk-sugar should be added to this unsweetened milk in the proportion of half a teaspoonful to 3 ounces of the diluted mixture. The unsweetened milk should be used within thirty-six hours of opening the tin; it decomposes much more rapidly than the sweetened milk, in which the cane-sugar acts as a preservative. Condensed milk has its value as a temporary food, where it is impossible to obtain fresh cow's milk, or where an infant is unable to digest ordinary diluted cow's milk. With an acid condensed milk gives a large firm clot, very like that formed in fresh milk, but, in spite of this, it is certainly more easily digested by some infants. If its use has to be continued several weeks the deficiency of fat should be corrected by the addition of ½-1 teaspoonful of fresh (48 per cent) cream to each 3-4 oz. feed, so as to bring the fat in the mixture to at least 3 per cent. The unsweetened milk should always be used in preference to the sweetened; but if only the sweetened is available (the other costs rather more, owing to its more rapid decomposition after opening, and therefore cannot always be used by the poor), it should be diluted freely, so as to reduce the sugar to 6-7 per cent, and then enriched by the addition of cream. It may also be necessary to give raw meat juice once or twice a day to correct the deficiency of proteids.

Condensed milk should not be used as the only food for an infant, except for very short periods. A very large proportion of the infants fed entirely or almost entirely on condensed milk develop rickets, partly, no doubt, owing to the excess of sugar, but perhaps more on account of the deficiency of fat. Moreover, in the process of condensation the milk has lost its antiscorbutic properties, and the use of condensed milk is a not uncommon factor in the etiology of infantile scurvy.

Proprietary Foods

The name of these is legion, and it would be impossible to describe them all in detail. There are few of them which are not occasionally of use, and still fewer which could not be usefully dispensed with. Almost all of them are preparations of cereals, and as such, almost all contain starch, but with this important difference, that while in some of them the starch has been partially or completely converted into the soluble carbohydrates, dextrin, maltose, or glucose, in others the starch remains unaltered. It is upon the degree of this conversion of the starch and upon the amount of fat present that the value of any individual preparation chiefly depends. None of the so-called "infant foods" which contain unconverted starch should be given to an infant under the age of six months; most infants are much better without any of these preparations until the age of nine months, and even then only those in which the starch has been partially converted should be allowed.

A very serious defect in all these "foods" is the low proportion of fat; some, indeed, scarcely contain any fat at all, and, prepared as they commonly are by the simple addition of water, they are totally unfit for infant food. In some of them this deficiency is to some extent corrected by giving them with diluted milk; but the fat value of the mixture is scarcely greater than that of the diluted milk alone, so that the addition of cream would be necessary to make the fat sufficient.

The scurvy-producing tendency of all these feeds is now well recognised, and, indeed, in the majority of the cases of pronounced infantile scurvy which have come under the writer's observation, the infants had been fed on one or other of these so-called "infant foods." Why they should produce scurvy is less evident; but this much seems clear, that the fault is a negative one rather than a positive: it would seem that in some way the food is "devitalised" in the process of manufacture, as milk is by sterilising, and to a lesser degree by boiling, and that the lack of this "vital element," if one may use such a vague term, is the cause of the scurvy. Here, again, the writer would like to emphasise the fact that weeks, perhaps months, before an infant shows any definite symptoms of scurvy, there may be a condition of cachexia and malnutrition which is as definitely related to the use of these sterilised "infant foods" as is the classical condition with spongy gums and subperiosteal hæmorrhages. If, therefore, any of these preparations are to be used for more than a few weeks, an adequate supply of fresh food of some sort should be given; and it may be pointed out in this connection that the addition of only a small quantity of milk, especially if it has been boiled, is not necessarily sufficient to prevent the development even of severe scurvy. The antiscorbutic power of milk is definite, but feeble; and if only small quantities of milk can be given, it may be necessary to supplement it with raw meat juice or lemon whey.

Such methods, however, are but unsatisfactory at the best, and the need for them should not be allowed to arise. There are very few cases in which any of these proprietary foods should be allowed to become the principal article of diet; and, in spite of all the advertisements, there is not a single "infant food" in the market which can adequately replace cow's milk as a food for infants.

With all their very serious faults, however, some of the proprietary foods have their value.

Dried milk preparations in particular are often well digested by infants who are quite unable to digest ordinary cow's milk; some of the best known of these are the Allenbury foods, No. 1 and No. 2, and Horlick's malted milk. These contain no starch, and when diluted with water according to the manufacturer's directions they contain respectively 2·6 per cent, 2·4 per cent, and 1·6 per cent of fat (the latter was obtained when four teaspoonfuls of Horlick's malted milk were added to four ounces of water). The curd produced by the addition of an acid to these preparations is much finer than in ordinary cow's milk, and seems to be easily digested. Such preparations are useful in some cases of gastro-intestinal disorder in infancy, but should only be used as temporary foods.

Another milk preparation is Nestlé's food, but this differs from the preceding in containing a considerable quantity of unconverted starch, together with some soluble carbohydrates; it is, in fact, a combination of a milk food and a partially malted cereal food. When diluted according to the directions it is very deficient in fat, containing considerably less than 1 per cent, and the presence of starch makes it wholly unsuitable for infants under the age of six months; after that age it may be useful given once or twice a day to accustom the infant gradually to a starch-containing food.

Completely malted cereal foods can be given at a much earlier age than those which contain unconverted starch. Perhaps the best known example of such a preparation is Mellin's food. This contains no starch, and consists chiefly of

soluble carbohydrates, together with a fair proportion of proteid. It contains only a trace of fat (·18 per cent), but as this food is always to be used with cow's milk the fat deficiency is less important. Such a preparation as this, which is intended as an addition to, not a substitute for, cow's milk, is much less open to objection than the other foods, and there can be no doubt that in some cases the addition of one teaspoonful or two of Mellin's food to one or two of the milk foods daily proves useful, not only by its own nutritive value, but also by facilitating the digestion of the milk. Mellin's food has, however, a laxative effect, which, although often useful, may contra-indicate its use where there is any looseness of the bowels. As already said, milk properly diluted is usually the only food an infant requires until the age of six or seven months, but where there is difficulty in digesting casein, especially if there is also troublesome constipation, and nutrition seems to be failing, a small quantity of Mellin's food may be given occasionally in the milk with advantage at as early an age as three months.

Partially malted cereal foods which contain some unconverted starch form the majority of the advertised "infants' foods." Such are the Allenbury No. 3 food and Savory & Moore's food. The latter, when mixed with a warm fluid, either water or milk, as directed, undergoes changes by which the starch is partially converted, owing to the presence of malt diastase in the dry preparation. Benger's food is somewhat similar, but the partial conversion of starch is accomplished by pancreatic ferments.

Such foods are very deficient in fat, and unless made with milk are wholly unsuitable for the feeding of infants; used with milk, however, they are sometimes of service during the later months of the first year. They should not be given with every feed, once or twice in the day is usually sufficient, starch being thus cautiously introduced into the diet.

Farinaceous foods, in which there has been little or no conversion of starch, such as Ridge's food, Neaves' food, Frame food, entire wheat flour, and the ordinary home-made arrowroot or corn-flour, and the various "biscuits," can seldom be taken with advantage earlier than the tenth month, and in most cases are better avoided until the end of the first year. They all have their use, and are valuable additions to diet in the second and later years. It is true that a certain number of infants can and do digest such foods as early as the seventh month, but these are the exceptions, and more often the result is flatulence and colic, if not more serious disturbance of digestion. The use of such foods during the first six months of life is a common cause of the pitiable condition of misery and marasmus which is so frequently seen amongst the infants of the poor.

The proportions of the more important constituents of some of the best known of the proprietary foods may be seen from the following table, which has been compiled from analyses published by various observers, several by Dr. Leeds:—

Composition of Proprietary Foods

	Allenbury No. 1.	Allenbury No. 2.	Horlick's Malted Milk.	Mellin.	Nestlé's Food.	Savory & Moore.	Allenbury No. 3.	Ridge's.
Starch	0·0	0·0	0·0	0·0	36·86	36·36	60·01	77·96
Soluble carbohydrates (Sugar, dextrin, etc.) .	65·48	69·02	63·59	68·18	40·9	44·83	25·1	5·19
Fat	13·15	12·48	8·4	0·18	4·25	0·43	1·05	0·63
Nitrogenous bodies . .	14·25	12·5	21·85	10·07	11·0	9·63	10·23	9·24
Ash	4·75	4·08	3·95	3·75	1·7	0·89	0·6	0·6

Eggs and Meat Foods

The value of eggs as a fat-containing food is perhaps hardly sufficiently appreciated. The yolk of an egg contains nearly 30 per cent of fat, and in this respect is a most desirable food for infants. As early as nine months of age an infant may have the yolk of an egg very lightly boiled once a day, and during the second year eggs are an important constituent in the dietary of infancy. The white of an egg is of value chiefly as a proteid-containing food, and is occasionally a useful addition to the diet where an infant is unable to digest the proteids of cow's milk, and is being fed with a mixture of cream and water, which, although containing a sufficiency of fat, contains very little proteid. The white of the egg must be given raw, and the most convenient way to give it is in the form of "albumin water."

The white of one raw egg is cut in several directions with a clean scissors and then mixed with half a pint of water, and flavoured with a little cinnamon water.

Given in this form the white of an egg is often very useful as the only food for twenty-four or forty-eight hours in cases of severe vomiting and diarrhœa.

Raw meat juice is used similarly to supply the proteid element where this cannot be taken in the form of cow's milk. Like the yolk of an egg, raw meat juice is rich also in iron and in phosphorus, and is specially valuable in rickets and anæmic conditions. An infant aged six months may take a teaspoonful of raw meat juice, either sweetened and given alone in a

spoon, or mixed with the diluted milk (after it is cool), or with cream and water, three or four times a day: to an infant of twelve months, two tablespoonfuls of raw meat juice may be given in the twenty-four hours.

Raw meat juice is made thus: scrape a quarter of a pound of lean raw beef into shreds in a cup; add to this two tablespoonfuls of cold water, and leave it, covered with a lid, to stand in a cool place for one hour; then strain it, and squeeze the juice out of the raw beef through muslin.

Meat broths are seldom to be recommended for healthy infants under the age of nine months, but in cases of troublesome vomiting or diarrhœa they may be given at the age of six months, or even earlier, as a temporary food. There are infants who seem unable to tolerate fat for a time, but who will take well a diet consisting chiefly of proteids; in such cases veal broth or chicken broth may be given with occasional feeds of whey, to which a little milk or cream may be added gradually, and in this way a return to a proper proportion of fat may by degrees be accomplished. Mutton broth, or, still better, veal or chicken broth, are to be preferred to beef tea, even when this is made very weak, for the feeding of infants; and, in the writer's opinion, the concentrated meat preparations which are now so widely sold are better avoided in the feeding of healthy infants. In cases where only very small quantities of food can be retained, and the infant is becoming exhausted by severe vomiting, a few drops of some concentrated meat preparation are often valuable as a temporary food.

Diet from Twelve to Eighteen Months

At the end of the first year an infant should have five or six meals in the twenty-four hours, the first being at 7 A.M., the last at 9 or 10 P.M. Cow's milk must still be the chief article of diet; but two or three feeds in the day may now consist of such starch-containing foods as finely crumbled stale bread soaked in milk or gravy; or a sponge-cake or rusk may be given sopped in milk. Most infants at this age like crumbled bread soaked in the fat from fried bacon, and it is a useful addition to the diet at this age; a few months later, thin slices of bread soaked or fried in the fat may be given. The yolk of an egg lightly boiled, or a custard made with egg, should be given occasionally, and once a day a little meat broth may take the place of milk. There is no objection at this age to such foods as Chapman's entire wheat flour, or Frame food, or a little well-boiled porridge once a day, and a thin slice of bread and butter may be allowed at two of the meals.

Diet from Eighteen Months to Two Years

After the age of eighteen months most infants require only four feeds in the day. At least a pint of milk should be taken in the twenty-four hours, but, in addition to the food already allowed, other articles may be given. Frame food or well-boiled oatmeal, with a drink of milk, will make a suitable breakfast, and for one meal in the day there is no objection to a tablespoonful of well-cooked and finely-mashed potato, which may be mixed with milk, or with the red gravy from underdone meat. Undoubtedly some infants can take potatoes without harm even earlier than this, but a very large number, not only of infants, but also of older children, suffer from disorder of digestion in consequence of being allowed potato too early or in too large quantity. It is a food which most children like, and which parents are only too ready to give, but it is quite unnecessary, and most children are far better without it until they are at least eighteen months old. There are few foods which are more often badly cooked than potatoes, and even when they are properly cooked they require to be finely mashed, not merely pressed through a fork, which leaves blocks of unmashed potato to worry the infant's digestion.

A tablespoonful of meat, very finely minced, or torn into fine shreds, may be given occasionally with the potato; underdone roast beef or mutton or the white meat from chicken or turkey, or a little boiled sole or turbot well pounded are most suitable, and the quantity may be increased gradually.

Milk puddings, rice, tapioca, sago, or semolina properly cooked may be given with a little juice from cooked fresh fruit or from prunes; junket or blanc-mange will also serve to vary the diet.

Bread and butter, rusks and sponge-cake, can be given more freely as the child approaches the end of the second year; but here a word of caution is necessary: whatever solid food is given, the child must be taught to eat slowly and masticate thoroughly; any one who has watched an infant cramming its food into its mouth, and bolting it with little or no attempt at mastication, can hardly wonder that indigestion is the result; nor is this fault limited to later infancy, older children are too often habitual food-bolters, and it may require patience and careful training to accustom them to take their food properly.

Lastly, it may not be out of place to mention the value of cold water. Many parents and nurses seem to be afraid to give an infant cold water to drink, as if it had some baneful effect; and, as a consequence, one sees, especially in hot weather, infants crying fretfully, not from hunger, but from thirst, and they are soothed at once by a drink of cold water. Pure cold water in small quantities—not, of course, to take the place of food, but to relieve thirst—may be given at any period of infancy; and nurses often need to be told that an infant who is ill, with a high temperature, should have the cold water that it craves.

Infanticide. *See* Medicine, Forensic (*Infanticide*).

Infantilism.—The persistence of the characters of childhood after the period of adolescence is reached, the individual so affected being a child physically and usually mentally also. *See* Cretinism.

Infarction.—The necrosed area produced by the blocking of the vessel which has supplied it with blood. *See* Embolism; Heart, Affections of Myocardium and Endocardium (*Effects of Cardiac Disease, Thrombosis, Embolism, and Infarction*); Liver, Diseases of (*Hepatic Infarcts*); Lungs, Gangrene of (*Etiology*); Lungs, Vascular Disorders (*Pulmonary Embolism*); Meninges of the Cerebrum (*Vascular Disturbances*); Pregnancy, Diseases of Placenta and Cord (*Pathological Infarction*).

Infection.

See also Alopecia (*Premature Alopecia, Etiology, Infective Fevers*); Aseptic Treatment of Wounds (*Wound Infection*); Athetosis (*Etiology, Infectious Diseases*); Breath (*Infection by*); Bronchi, Bronchitis (*Acute Bronchitis, Etiology, Infective Causes*); Disinfection; Hysteria (*Etiology, Infection*); Immunity; Leucocytosis; Micro-Organisms; Mumps; Myiasis; Pregnancy, Intra-uterine Diseases (*Infectious Fevers*); Respiration (*Bacteriological Aspects*); Skin, Bacteriology of; Tonsils, Diseases of (*General Considerations*).

By "infection" is understood the introduction into the body of a pathogenic micro-organism which is capable of multiplying within it. Diseases, therefore, which depend on such an infection are called "infective" or "infectious" diseases. For a long time it was the custom to draw distinctions between infection and contagion, but, broadly speaking, the process is the same in every case, and all so-called contagious diseases are certainly infectious in the modern sense of the term. It is true, of course, that certain infections are only contracted by direct contact, as for instance syphilis, rabies, and gonorrhœa. But others, such as anthrax and small-pox, can be contracted not only by direct contact, but also through the intermediary of the air, and are thus, to use the old terms, both contagious and infectious. It is more convenient, therefore, to class all diseases which depend on the entrance of a living organism into the body as "infectious."

The living organisms which cause these diseases are nearly all vegetable in their nature, and are of the class "fungi." The vast majority of them are bacteria. But besides these vegetable organisms certain protozoa may give rise to infectious diseases, the most notable instance being the spirillum of relapsing fever. This is not the place to discuss or open up the whole question of bacteriology. It is sufficient to note that bacteria are divided into two main groups, that of (*a*) parasites, which can exist and thrive in living animal tissues, and that of (*b*) saprophytes, which can live outside living animal tissues, on dead animal or vegetable matter, or on inorganic substances. The first group, again, may be subdivided into those germs which cannot exist outside living animal tissue, "obligatory parasites," and those which can do so for a longer or shorter period, "facultative saprophytes." And the same subdivision may be made of the second group into "obligatory saprophytes" and "facultative parasites."

Each micro-organism, whether it be a bacillus or some variety of coccus, has the power when growing on the living tissues or in suitable media of manufacturing a toxine which is peculiar to itself. In an infectious disease the majority of its distinguishing symptoms are due to the toxine rather than to the presence of the bacillus itself, although the latter may in certain diseases be the cause of local conditions which may ultimately cause the death of the infected person. How the toxine itself is produced opens up a question of the greatest obscurity. Some hold that it is directly derived from the cell plasma of the bacillus, and shares the specific qualities of that bacillus. Others, again, regard it as the result of the action of the bacillus on proteid substances.

The micro-organism is introduced into the human body in a variety of ways. It may be in the first place directly inoculated. It may be inhaled. It may be ingested. In order that it should be introduced in one of these ways, it must either contaminate various articles with which the person attacked is in contact, or else be carried in the air, or, lastly, contaminate food or water. The germs of some diseases, such as small-pox and measles, are probably in most cases inhaled; that of typhoid fever, on the other hand, is always ingested. It is possible, however, that germs which are primarily inhaled may find their way from the mouth and pharynx to the alimentary canal. This would give an easy explanation of those cases of typhoid which seem to be due to dust. The action of insects in assisting the spread of infection depends on their contaminating food. (*See* article "Disinfection," vol. ii.)

After obtaining an entrance to the body, the germ apparently lies more or less latent for a time before it gives off any marked amount of toxines. This latent period, the period of *incubation*, distinguishes an infection from an intoxication, no such latent interval occurring in the case of

the latter. This period may be long or short. Various infectious diseases show a range from a few hours up to nearly four weeks, some indeed much longer. Moreover, the period of latency is by no means constant in the case of any given disease, the variation, for instance, in typhoid fever being nearly three weeks. It would appear that in some diseases, at least, the length of the incubation period may be affected by the dose of infection which the subject has undergone. The larger the dose the earlier the disease may begin.

It must always be remembered, in computing the length of an incubation period, that the reckoning must be made from the exposure to infection to the first rise of temperature. Needless confusion is sometimes caused by dating up to the time when the rash of an exanthematous fever appears. Again, it must be remembered in cases where the latent period has appeared unduly long that it is perfectly possible that infective matter may have been carried about on the clothes or elsewhere by the patient, and that the real date of infection was perhaps a week subsequent to the last exposure.

When an infection has occurred it may be local or general. In the first case, as for instance diphtheria or tetanus, the germs obtain a nidus at a given spot and cause some local disturbance and inflammation. They are not found in the blood, and the constitutional disturbance set up is due to their toxines and not to themselves. On the other hand, in a disease like anthrax the bacilli are found in the blood-vessels and the infection is general. Not only are the toxines in such cases given off probably more rapidly, but the mere mechanical presence of the bacilli may cause lesions of great importance, such as emboli.

Again, an infection may be specific or non-specific. In many diseases we know that the germ with which they are identified always causes an exactly similar series of symptoms and of pathological lesions. Such infections are truly specific. But there are other diseases which are due to micro-organisms, and so must be classed as infective, which present the same clinical and pathological features, and yet may be found in different instances to be due to perfectly distinct germs. Thus pneumonia may be due to more than one organism, and the same holds true of ulcerative endocarditis. Suppuration, again, may be caused by quite a number of different micro-organisms. (*See* "Micro-organisms" and "Suppuration.")

The exact progress of an infection when once the germs have obtained an entrance into the body remains a question for speculation. No doubt a certain number of them are destroyed by the phagocyte cells, but these cells are probably without action on the toxines given off by the bacteria. The sudden manner in which many of the acute infections start would lead one to believe that the body, by means of its cells and fluids, can offer resistance up to a certain point by counteracting in some way or other the multiplication and activity of the invading germs.[1] After a longer or shorter interval the balance between the attack and defence is suddenly lost and the symptoms of the infection suddenly appear. As the infection progresses, various substances are elaborated by the resisting organism which possess germicidal and antitoxic properties, and the invading germs are by degrees, or in some diseases where there is a well-marked crisis suddenly, killed off or rendered harmless and their toxines are neutralised. In some cases, however, the reaction of the infected individual may be below the average, or, on the other hand, the infection may be more severe, and the toxines not being neutralised the individual succumbs to toxæmia and dies. It must be remembered that death may also occur from lesions caused by the presence of the bacilli, as for instance death by suffocation in laryngeal diphtheria, or by hæmorrhage or perforation in typhoid fever.

The amount of "resistance" offered to various infections by different individuals may vary considerably; that is to say, there may be an actual *predisposition* on the part of the individual to certain infections. This predisposition may be "natural,"—that is to say, that certain species of animals and certain races of men are particularly prone to develop certain infections,—that is to say, their natural resistance towards these infections is below the normal. On the other hand, predisposition may be artificial or acquired. Many diseases are more easily contracted by persons whose power of resistance has been weakened by starvation, fatigue, loss of blood, damaged digestive power, or exposure. In such cases the vitality of the individual is so depressed that germs which would find no chance of obtaining a lodgment in the tissues in a state of health, easily succeed in infecting him.

On the other hand, as opposed to predisposition, there may be *immunity* to an infection. This immunity, also, may be either natural or acquired. Natural immunity is shown in the cases of individuals who, frequently exposed to an infection, fail to contract it. It may be in all probability inherited. The fact that the father and ancestors of a person have all suffered from a given infection may act as a protection against the child contracting that infection badly or contracting it at all. The converse of this is seen in the fatal ravages caused by such a disease as measles, when it is introduced into a community in which it was previously unknown. Epidemics in the Fiji and in the Faroe Islands are instances of how serious a comparatively mild disease may be when it obtains a perfectly new soil. The immunity of an animal to

[1] See "Immunity," p. 342.

infection may be complete, that is to say, certain species appear incapable of contracting certain infections, or it may be partial, in which case the power of resistance to a certain infection appears increased, and the disease, if caught at all, runs a mild course. This natural immunity has been variously attributed to (α) phagocytosis, (β) to the presence in the blood of certain germicidal bodies called "alexins," which are of a different nature from antitoxins, and which can be separated from the blood and tissues of most animals, and (γ) to a vital reaction of the tissues at present unexplained. This subject is fully discussed in the article "Immunity."

Immunity may be "acquired" by a previous attack of the same infection. That is to say, one attack of an infectious disease usually protects against that disease for life, or at least for a considerable period. This immunity is of course acquired during the attack of the infection, and it is its establishment that leads to a favourable termination. It may also be conferred by vaccination or by the injection of antitoxin prepared from the serum of an immunised animal. Such antitoxin may be not only prophylactic in its nature, but actually curative, and it is on this that treatment by serum is based.

It must be remembered that an infection is not always due to one germ only. There may exist what are called "associated infections," where the illness of the patient is due to the presence and action of two or more species of micro-organisms in combination. Thus clinical diphtheria may be not merely the infection of a mucous membrane by Löffler's bacillus. With that bacillus may be associated various other forms of micro-organisms, such as, notably, the staphylococcus or the streptococcus. And a throat which is infected by these septic organisms is very often the starting-point of a toxic infection more severe than when merely a pure culture of the bacillus is present. In fact the more severe cases of diphtheria are those associated with other micro-organisms. In the same way in inoculation experiments on animals it is found that certain microbes are far more virulent in their specific action if other micro-organisms are injected with them.

Further, two or more pathogenic germs can exist in the human subject concurrently, and independently of each other produce the characteristic lesions and symptoms associated with each. In other words, *coexistence of infectious diseases* is quite possible, and, although it is a possibility which is too often disregarded, is comparatively common. The old view that two infections cannot exist together is quite an error, and any one with any experience of infectious disease has seen scores of cases of concurrence. One of the most frequent is the concurrence of scarlatina and diphtheria. The two diseases may exist together from the same date, or one may follow closely or tardily on the other. Again, a combination of scarlatina and measles in the same patient is by no means rare. And cases have been noted in which scarlatina, diphtheria, measles, and varicella have practically coexisted.

The Prophylaxis of Infection.—To combat infection the most efficient method is care of the general sanitation in the first place, and of the health of the individual in the second. Remembering the various ways in which infection may be carried to the individual, it is the duty of the community to secure that he should live in a pure atmosphere, have a water and milk supply that is above suspicion, and that the cleanliness of streets and houses should be made an object of rigid care. Should infection break out, the local authority depends on, firstly, *notification*, a system under which every practitioner who diagnoses a case of infectious disease is bound to notify the medical officer of health, which enables that officer to inquire into the cause of the outbreak and take the necessary measures for preventing its spread. Secondly, it depends on *isolation* of the infected person either in a fever hospital or in suitable rooms in the patient's own home. The latter plan is seldom safe unless the house is large and airy, and the whole top flat can be placed at the disposal of the invalid. Thirdly, in certain more dangerous diseases, such as plague or typhus, *quarantine* may be imposed on persons who have been in contact with the sufferer. Fourthly, in some diseases *protective inoculation*, such as antitoxin in diphtheria, or vaccination in small-pox, may be offered to the so-called "contacts." In addition to these steps great attention must be paid to *disinfection*, and the reader is here referred to the article under that head (vol. ii).

For the benefit of medical officers of schools the following rules will be found useful :—

Rules for the Prevention of Infectious Disease

1. The following diseases are considered infectious :—Whooping cough, diphtheria, measles, scarlet fever, German measles, small-pox, typhus fever, typhoid fever, chicken-pox, mumps, ringworm.

2. At the beginning of each term, certificates, which will be duly sent to parents and guardians, are required to be shown to the headmaster on the entry or return of pupils, signed by the parents or guardians, not earlier than the day before admission, stating that to the best of their knowledge the pupil has not, *for at least three weeks*, been exposed to any infectious disease, nor entered any house where such disease has existed.

3. If a pupil take an infectious complaint, or has been in any way exposed to infection *during*

the holidays, parents or guardians must refer to this list of Rules, and if the case come within their scope must communicate with the headmaster.

4. If a pupil take an infectious complaint, or has been in any way exposed to infection *during the term*, notice must at once be sent by the parents or guardians to the headmaster.

5. With regard to Rule 2, no pupil will be allowed to enter or return to school without the certificate signed by *parent* or *guardian*. In the case of a pupil who comes under Rule 3 or 4, he will not be allowed to enter or return to school without a printed form, signed by a *doctor*, to the effect that the said Rules have been complied with.

6. Periods of absence for pupils who have been suffering from any of the above diseases :—

Whooping-Cough.—Six weeks from the commencement of the whooping, provided the characteristic spasmodic cough and the whooping have ceased.

Diphtheria.—In not less than three weeks, when convalescence is completed,—there being no longer any form of sore throat, nor any kind of discharge from the throat, nose, eyes, etc., and no albuminuria.

Measles.—In not less than three weeks from the appearance of the rash, if all desquamation and cough have ceased.

Scarlet Fever.—In not less than six weeks from the appearance of the rash, if desquamation have completely ceased, and if there be no discharge from the nose, ears, glands, or any suppurating sore.

German Measles.—In not less than two weeks from the appearance of the rash.

Small-pox.—One week after the crusts have all separated.

Typhus Fever.—When convalescence is complete, five weeks.

Typhoid Fever.—In not less than six weeks from the commencement of the disease, or of a relapse, if such occur.

Chicken-pox.—One week after the crusts have all separated.

Mumps.—Three weeks from the beginning of the swelling, if all complications have subsided.

Ringworm.—(*a*) *Of Body*—One week after the disappearance of the characteristic eruption. (*b*) *Of Scalp*—One month after the pupil has been certified by a medical man to be apparently free of the disease. The pupil must also be re-examined within two days of return to school, and bring with him a certificate that he is still free of the disease.

In addition to these various periods the patient must in all cases be thoroughly disinfected (*see* vol. ii. p. 411).

7. In the case of a pupil being exposed to infection, the following quarantine times may be considered safe if *thorough* disinfection be carried out :—

Whooping-Cough	21	days' quarantine.
Diphtheria	10	,, ,,
Measles	16	,, ,,
Scarlet Fever	10	,, ,,
German Measles	21	,, ,,
Small-pox	18	,, ,,
Typhus Fever	21	,, ,,
Typhoid Fever	21	,, ,,
Chicken-pox	20	,, ,,
Mumps	26	,, ,,

Ringworm :—(*a*) A pupil who has been casually exposed to the infection of ringworm must be kept at home and carefully examined daily for ten days before being sent to school. Should any suspicious spot appear during this time medical advice must be taken before his return. (*b*) No pupil shall attend school from a house in which there is a case of ringworm *of the scalp* within a fortnight of the detection of the disease, and then can only attend if he brings with him a medical certificate that he is free from ring-worm himself, and that he has been scrupulously isolated during this time from the case under treatment.

N.B.—As the risk of infection in this variety of ringworm is much less in the case of older pupils (say over 15), special exceptions may be made in their case on communication with the headmaster or headmistress.

8. The fact of a pupil's having had any of the above diseases does not exempt from the above periods of quarantine, except in cases of whooping-cough, German measles, chicken-pox, or mumps, in which cases a pupil may enter or return to school after one day's quarantine.

9. No pupil shall enter or return to school from a house in which there has been any person suffering or convalescent from any of the above infectious diseases, until such house has been thoroughly disinfected to the satisfaction of the medical man in attendance ; and it must be borne in mind that the quarantine must date from the time that such disinfection was completed.

10. No article of any kind which has been worn or used by any person suffering from infectious disease, or has been in contact with any such patient or his attendant, shall be brought to school, unless it has been dealt with in accordance with the suggestions for disinfection given (*q.v.*).

Note.—Influenza must be considered an infectious disease, but as yet no definite rules regarding the length of quarantine and of the infective period are formulated. Meanwhile it is recommended that any pupil who has had influenza should be thoroughly convalescent before returning to school ; and that pupils from a house in which there is a case of influenza should be isolated from the patient, and should be kept at home if they show the slightest sign of illness.

Infiltration.—The deposition within and diffusion throughout an organ or tissue of substances foreign to that organ or tissue brought to it generally in the blood stream. *See* Fatty Infiltration; Inflammation; etc.

Infirmary. *See* Hospitals.

Inflammation.

See also Appendix Vermiformis, Appendicitis; Arteries, Diseases of (*Arteritis, etc.*); Atrophy (*Result of Inflammation*); Auditory Nerve and Labyrinth (*Inflammation of the Labyrinth, Acute and Chronic*); Balneology (*General, Use of Baths in the Results of Inflammation*); Bladder, Injuries and Diseases of (*Cystitis*); Bladder, Injuries and Diseases of (*Female Bladder, Cystitis*); Bone, Diseases of (*Pyogenic Diseases*); Brain, Inflammations; Bronchi, Bronchitis; Choroid, Diseases of (*Choroiditis*); Colon, Diseases of (*Inflammation*); Conjunctiva, Diseases of (*Inflammatory Affections*); Dermatitis Herpetiformis; Diaphragm (*Inflammation*); Duodenum (*Inflammations*); Dysentery; Ear, External, Diseases of (*Otitis*); Ear, Acute Inflammation of Middle Ear; Ear, Middle, Chronic Suppuration; Ear, Middle, Chronic Non-Suppurative Inflammation; Erythema; Fallopian Tubes (*Inflammation*); Gall-Bladder and Bile-Ducts, Diseases of (*Cholecystitis, etc.*); Heart, Affections of Myocardium and Endocardium (*Endocarditis, etc.*); Intestines, Diseases of (*Enteritis*); Iris and Ciliary Bodies (*Inflammatory Affections*); Joints, Diseases of (*Synovitis, etc.*); Lacrymal Apparatus, Diseases of (*Diseases of Lacrymal Sac*); Larynx, Acute and Chronic Inflammations; Leucocytosis; Liver, Diseases of (*Cirrhosis*); Mammary Gland, Diseases of (*Inflammatory Affections*); Mediastinum (*Mediastinitis*); Meninges of the Cerebrum (*Inflammations*); Muscles, Diseases of (*Inflammatory*); Nephritis; Œsophagus, Injuries and Diseases of (*Inflammation*); Ovaries, Diseases of (*Inflammation*); Pancreas, Diseases of (*Pancreatitis*); Parotid Gland (*Inflammatory Conditions*); Pericardium, Diseases of (*Pericarditis*); Peritoneum (*Peritonitis*); Pharynx, Acute and Chronic Pharyngitis; Pleura, Diseases of (*Pleurisy*); Pneumonia; Prostate Gland (*Inflammation*); Rectum, Diseases of (*Ulceration*); Retina and Optic Nerve (*Inflammation*); Scrotum and Testicle, Diseases of (*Orchitis, Epididymitis*); Stomach and Duodenum, Diseases of (*Inflammation, Gastritis*); Teeth (*Periodontitis, etc.*); Thyroid Gland, Medical (*Thyroiditis*); Tongue (*Inflammatory Affections*); Tonsils, Diseases of (*Tonsillitis, Acute, Chronic*); Urethra, Diseases of (*Inflammation*); Uterus, Inflammations of the; Vesiculæ Seminales (*Inflammation*).

Since the introduction of the term inflammation to indicate the cardinal symptoms of redness (rubor), swelling (tumo), heat (calor), and pain (dolor), which characterise the inflammatory processes when seen upon the skin or other similarly exposed part, its meaning has undergone so many additions and modifications, that it is difficult to find any two pathologists agreeing upon its significance. A definition which would be satisfactory to all is impossible, and so much at variance are their views as to its nature, that unity of opinion is not likely ever to be attained. Some would restrict it to mean only a local disturbance of nutrition accompanied by exudation from the blood-vessels, while others would extend it to include so many other changes that it becomes practically synonymous with local disease. Owing to the differences of opinion being so wide and practically irreconcilable, it is thought by many that the term inflammation should be discarded from pathological nomenclature. This view is probably scientifically correct, but it is not likely to prevail. So much valuable work has been done under the time-honoured title of inflammation, and so rich is it as a storehouse of knowledge, that it will not be easy, even if it were wise, to displace it from its high position in pathological literature. In its earlier and simpler acceptations it includes a series of phenomena of pre-eminent importance in the study of many diseases pretty generally recognised and understood by pathologists. It is important to note at once that it is not a simple vital process, but a combination of different vital processes which may be conveniently studied and demonstrated by an experiment similar to that originally performed by Cohnheim upon the web of the frog's foot. As modified by Coats, it is so easy of performance and its phenomena so readily observed, that it has long been with teachers the favourite method of introducing the subject to their students. The frog should be pithed and curarised. Its foot, with the web spread out, is fixed upon an ordinary microscopic glass slide, so that it may be examined under the microscope. A small portion of the surface of the web is snipped off with scissors, just deep enough to remove the epithelial layers without drawing blood, and the subsequent inflammatory processes carefully observed. They are as follows: 1. Active Hyperæmia and Acceleration.—The arteries in the immediate neighbour-

hood of the injured part dilate, then the veins, and the rate of blood-flow therein is obviously accelerated. These changes last a considerable time, and are followed by others generally in about an hour. Many capillaries formerly invisible begin to appear and become distended with blood. 2. Hyperæmia and Retardation.—After a time the rate of blood-flow becomes slower, and continues to do so until it is slower than normal. The vessels, particularly the capillaries and veins, instead of showing the usual well-marked distinction between axial stream of corpuscles and peripheral stream of plasma free from cells, show a mixing of these streams. The axial one broadens out and gradually encroaches upon the peripheral one. Then leucocytes fall out into this peripheral stream and move slowly along, every now and then becoming adherent for a moment to the vessel wall only to move slowly onwards again at intervals. They may even regain the axial stream. Blood-plates often also appear in this marginal zone. The blood-flow gradually becomes still slower, and red blood corpuscles pass out into this same zone, and soon all distinction between axial and peripheral streams is lost. The lumen is filled with a mass of red and white corpuscles whose shape and outlines are now distinctly seen. The latter gain the wall of the vessel in increasing numbers, and adhere firmly to it, generally becoming pear-shaped owing to the pressure of the blood-flow. The slowing continues until the blood is moved onwards merely by a succession of jerks, or even oscillates slowly backwards and forwards, ultimately coming to a complete standstill, a condition of stasis. 3. Exudation of Fluid.—A considerable oozing of clear fluid now comes from the wound. It began earlier, but has now become marked. It comes from the distended vessels, particularly the capillaries and veins, and is generally spoken of as an outpouring of lymph. 4. Escape of Blood Corpuscles.—The leucocytes adherent to the vessel walls gradually make their way through until they lie altogether outside the vessels. It takes a leucocyte a very considerable time to emigrate from a vessel, probably because it has to squeeze itself through an aperture greatly smaller than itself. When half through it may be seen as a rounded swelling both outside and inside the vessel, connected by a very narrow thread. Emigration occurs most abundantly from the small veins, but it also occurs to some extent from the capillaries. Although the wall of the latter is so much thinner, yet the lumen is so narrow that the same margination and accumulation of leucocytes cannot take place. The process generally continues steadily for several hours, until a great concourse of leucocytes showing active amœboid movement can be seen lining the outside of the veins and in lesser numbers the capillaries. Little groups of red blood cells may also be seen in places among the leucocytes. The passage of the red cells through the vessel walls is known as diapedesis, a term which has been extended by some authorities to include also the emigration of the leucocytes. Many of the latter move from the neighbourhood of the blood-vessels towards the injured surface, and they may escape in such numbers in the exudate that it becomes opaque instead of clear. Others break down, and fibrin appears in the exudate, so that the gap becomes filled with a colourless clot which overlaps the edges. 5. Reaction of the Fixed Tissue Cells.—The epithelial cells at the margin now begin to proliferate and grow over the wound towards one another. Circulation begins afresh in the affected vessels, slowly at first, but steadily and gradually. The vessels contract, the leucocytes disappear, and soon (generally in about three days) the circulation appears to be perfectly restored to its normal state. The proliferation of the epithelium has meanwhile continued and is completed about the same time, so that now all traces of the injury have disappeared. These later stages cannot be seen in the pithed frog, and hence fresh animals must be used; and further, the greatest care must be taken to protect the wound from all contamination. The mesentery or tongue of the frog may be chosen for experimentation instead of the web. The mesentery of the cat, dog, etc., or the ear of the white rabbit, or wing membrane of the bat, are also suitable structures, as they all permit the direct observation of the circulation under the microscope. Exactly similar changes are seen in naturally occurring inflammations of the skin and serous surfaces, such as the peritoneum, pleura, meninges, and joint surfaces, etc.

The Causes of Inflammation.—The exciting cause of the inflammation in the foregoing experiment is a mechanical one. Chemical, thermic, and electrical influences act in a similar manner. Thus the mere exposure and fixation to the microscopic slide of the mesentery of a warm-blooded animal is sufficient to set up a severe inflammation. The rapid cooling and evaporation must be held in check otherwise necrosis may set in. The mechanical, thermic, chemical, and electrical agents are classed as external noxæ, and to them must be added the most important of all, viz. infections in which bacteria are the causal factors. A combination of noxæ may occur. Thus bacteria may gain a footing in a tissue already inflamed through the action of another agent. The mechanical, electrical, and thermic noxæ must always act externally, causing inflammation at their site of impact, whereas bacteria may enter the body without setting up any change at their point of access, and being transported by the blood or lymph may cause inflammations at other and distant parts of the body. Such inflammations are metastatic, and the noxæ may

be called internal. Bacteria act through the agency of their products (chemical substances) technically known as toxines. They may be formed by bacteria within the body, or may be performed and introduced as such into the body, *e.g.* in poisonous tinned meats. It is to be remembered also that other injurious chemical substances may be produced within the body entirely independently of bacteria, which may act in the same way. This may occur in functional disturbances of several of the internal organs, such as the liver, kidney, intestines, etc. These abnormal products of metastasis may be absorbed by the lymph or blood, and set up metastatic inflammations exactly as the toxines of bacteria do. Any noxa, external or internal, in order to cause inflammation must be of a certain degree of intensity. If too severe it will lead to the death of the tissue, and then vital processes are no longer possible.

The Phenomena of Inflammation. — The ordinary processes of inflammation as seen in the foregoing experiment and similar lesions may be arranged as follows: 1. Active hyperæmia and acceleration; 2. Hyperæmia and retardation; 3. Escape of fluid; 4. Escape of blood corpuscles; 5. Changes in the fixed tissue cells. The cardinal symptoms of redness, heat, swelling and pain are appreciable by the unaided senses, and are easy of explanation. The redness is due to the greater amount of blood in the part, the heat to the rapid transference of the hotter internal blood to the surface, and an increased flow of blood through the part. It is only seen in external, not in internal inflammations. The swelling is due to the distension of the blood-vessels and the exudation; the pain (to which functional disturbance may be added) to the increased pressure upon the nerve endings, to the impeded circulation and nutrition, and partly also directly to the exciting cause or noxa. A closer examination aided by the microscope reveals the other changes just mentioned which are invisible to the naked eye, and include these coarser microscopic changes. They may be studied in detail.

I. *Active Hyperæmia and Acceleration.*—The dilatation of the arteries is brought about partly reflexly either by a paresis of the vaso-constrictor nerves or a stimulation of the vaso-dilators, and partly by a direct paralysing action on the arterial and capillary walls and the connective tissues surrounding them. It may pass off without the appearance of other inflammatory phenomena, and the latter may appear without its preceding them. Hence it is not an indispensable process, but a more or less fortuitous forerunner of inflammation.

II. *Hyperæmia and Retardation.*—The dilatation of the veins and capillaries follows upon that of the arteries, and increases. Retardation of the blood-current follows upon the acceleration. The cause is the alteration of the vessel walls and surrounding tissues, produced by the action of the noxæ, especially those tissues in the neighbourhood of the capillaries. These changes will be presently described under the reaction of the fixed tissues. They lead to increased friction between the blood and the vascular walls, so that the red and white corpuscles escape into the peripheral stream. The blood-plates, which may also fall out, are held by some to be normal constituents of the blood, and by others to come from degeneration and breaking down of the red cells. These processes are practically always observed in all inflammations of vascular tissues, and hence may be regarded as essential phenomena of such inflammations.

III. *Escape of Fluid.*—There is a liquid transudation from the blood-vessels similar to the normal lymph transudation, but the fluid differs in amount and composition from the lymph. It is more abundant, more albuminous, and contains a greater amount of solids. A deposit of fibrin sometimes occurs. The cause of these differences is the alteration brought about in the vascular walls through the action of the noxa. The normal selective power of the vascular walls is not lost, for the altered walls still act as a secreting and not as a mere filtering apparatus. The alterations in the blood-pressure may greatly assist the transudation process, but the alterations in the vascular walls are the primary cause. The exudate infiltrates the fissures of the surrounding tissues and may cause considerable swelling. When it finds its way on to a free surface, such as a mucous or serous surface, it may escape or collect in a serous cavity. Part of it may be absorbed by the cells and other tissues, which may consequently show coarse granulation, mucoid, and other degenerations. They may even undergo complete disintegration, and their elementary constituents may dissolve in the exudate. The chemical composition of the fundamental substances in the exudate may also undergo change through their contact with the tissues of the part. The exudate always contains cells, and if it be very rich in leucocytes it is called pus. It may also contain large numbers of red corpuscles when it is spoken of as hæmorrhagic. The exudate may be taken as a measure of the severity of the inflammation, for usually the greater the severity the greater the exudate, and the more prolonged will the inflammation be. Its amount and character, therefore, depends largely upon the nature and degree of intensity of the noxa, but also partly upon the region affected and the condition of the tissues. It is an essential phenomenon of inflammation in vascular tissues, and is never really absent therefrom. While often a useful process, it is by no means always so. When serous it may act a beneficial part by flushing out the injured area and diluting the noxa. This will be advan-

tageous, especially upon external surfaces and mucous membranes, as a large quantity of serous fluid may wash away the poisons or at least weaken their effects. When fibrinous it may circumscribe the inflammation by preventing the spread of the irritant. This is well seen in the fibrinous adhesions which so frequently localise an appendicitis or a pelvic peritonitis, and even in more diffuse forms, such as pericarditis, it may do good by closing the stomata and interfering with toxine absorption by the lymphatics and blood-vessels. The exudate may also contain bactericidal and digestive substances, leading to the production of peptones and the death of bacteria. It may lead to increased nutrition of the affected region, which may be useful in the later, though rarely in the earlier stages of inflammation. On the other hand, the exudate is positively hurtful when it collects in the body cavities and ventricles of the brain, and when it accumulates in the lung alveoli, or in the pharynx and air passages. Further, instead of proving injurious to bacteria, it may be actually helpful by providing them with a good nidus for their further growth and development.

IV. *The Escape of Blood Corpuscles.*—1. The Leucocytes.—Their escape from the blood-vessels is an active vital process. The first step is their margination along the wall of the blood-vessels, especially the small veins. It is brought about by action of the noxa upon the vessel walls, as it never occurs in a healthy vessel. It is a deteriorative or degenerative action, generally demonstrable histologically. The nucleus of the endothelial and other cells becomes rounded, the protoplasm granular or vacuolated, or mucoid, colloid, or other degeneration appears. Even in the exceptional cases in which there is no detectable histological change there is always a disturbance of the perfect endothelial pavementation of the lumen. The cement substance between the endothelial cells is deranged and loosened. It becomes irregularly distributed along the lumen and widened in places, as can be shown by treating it with the silver nitrate process. The amount and degree of this change will depend mainly upon the nature and intensity of the noxa, but it is rarely so great as to cause the appearance of actual visible pores. They exist potentially, and the leucocytes, after margination, become adherent to the endothelial cells, and commence to push a part of their protoplasm through between them at intervals here and there along the vessel wall, and the entire cell body gradually follows. In the capillaries, where the process can be most easily studied on account of the thinness of their walls, the leucocytes are always seen to pass out between and never through the bodies of the endothelial cells. It is chiefly the polymorpho-nuclear leucocytes, comprising roughly 70 per cent of the total number of the corpuscles present in the blood, which emigrate. They pass out of the vessels through their amœboid movement, and may in the same way transport themselves to considerable distances. The activity of this amœboid movement appears to be largely dependent upon the nature and intensity of the noxa. The more the blood-vessel wall is deranged the more readily can it occur. They do not show it while still within the vessels and previous to adhesion to their walls. It has been suggested that substances are formed in the tissues in inflammation which attract the leucocytes, and the term positive chemotaxis has been introduced to designate this attraction. Many substances can be experimentally shown to exert it, *e.g.* thin solutions of glue, of the gluten of wheat, and liquids containing the products of bacterial action. Substances having this positive chemotaxic action may be produced from various sources in inflammation, such as the products of bacteria and the products of degeneration of the fixed tissues. The margination and adhesion of the leucocytes are apparently entirely due to the degenerative changes in the vessel walls, uninfluenced by chemotaxis. Nor can the latter be regarded as the cause of either the beginning or the continuance of the amœboid movement, since leucocyte emigration may take place when it is certain that no such action is being exerted. It cannot be more, then, than an active and important stimulant thereof. Certain substances may arrest or prevent the emigration of leucocytes, *e.g.* solutions of quinine, iodoform, salicylic acid, and a $1\frac{1}{2}$ per cent solution of common salt. This action is capable of different explanations. It may be attributed to a negative instead of a positive chemotaxis, causing an inhibition of amœboid movement, or to a contraction of the vessel walls which interferes with their permeability and with the adhesion of leucocytes. Such substances are not, therefore, suitable washes or applications for wounds where free leucocyte emigration is desired. Leucocyte emigration occurs most abundantly in inflammations set up by the pyogenic bacteria, viz. the staphylococci and streptococci, which cause what are known as suppurative inflammations or suppurations. They have been most carefully studied in their experimental production in the skin and cornea, and in their natural occurrence in various organs and tissues. The leucocytes emigrate extensively from the blood-vessels, and collect chiefly around and among the cocci. Many of them have their protoplasm crowded with cocci which they have ingested. This engulfing of the cocci by the leucocytes, which are consequently called phagocytes, is an active process, and is held by Metschnikoff and his followers to be of vital importance in controlling, curbing, and counteracting the pyogenic inflammatory processes, by gradually bringing about the destruction of the causal bacteria. This is the doctrine of phagocytosis. It may be

carefully studied in the experimental production of abscesses. There is first an accumulation of lymphocytes in the affected area, and then emigration of polymorpho-nuclear leucocytes from the neighbouring blood-vessels occurs. They collect around the cocci, which are meanwhile multiplying rapidly, and being largely ingested by many of the leucocytes. The liquid exudate from the blood-vessels collects in the tissue spaces, and may help the excretions of the cocci (the main agents) to digest and liquefy the formed tissues of the part. Upon a free surface the liquefied tissues escape and leave an ulcer. In a deep-seated part they collect as a liquid mass containing cocci, degenerated leucocytes, and tissue debris, and form an abscess. The liquid contents are called pus. Bacteria are generally present in acutely formed abscesses, but often absent, having disappeared in chronic ones. They are always seen in greatest numbers at the margins of the abscess, and it is here also that phagocytosis is most active; and further, so long as the suppurative process is extending, the cocci are observed in the tissue spaces outside and beyond the leucocytes, whereas the reverse is the case when it has ceased to spread. It is the polymorpho-nuclear and the large hyaline uninuclear leucocytes which act as phagocytes. It is generally held that the coarsely granular eosinophile leucocytes, which are present in very small numbers in the normal blood, but plentifully in the tissue spaces, never act as phagocytes. The writer, has, however, seen them crammed with gonococci in gonorrhœal discharge. The bacteria ingested by these various phagocytes can in many cases be shown to be still active and virulent, and capable if liberated of exerting their specific action, while in other cases they are degenerated and inert, and dying or dead. Relying upon this and similar arguments, and particularly upon the reactions to irritation exhibited by leucocytes in the lower animal structures, such as the tadpole's tail and tissues of the invertebrata generally, the opinion that phagocytosis is always a beneficial process has received much support from pathologists especially in recent times. It has been likened to a struggle between the leucocytes and bacteria, in which both strive for the mastery. If the former should prove victorious the disease will be arrested and cured, and *vice versa*, the disease will continue and extend. This doctrine can no longer be upheld. It is undoubtedly true that phagocytosis may be a beneficial process. This is seen in many of the acute suppurative inflammations caused by the pyogenic cocci, and in other instances where the leucocytes ingest and destroy active and vigorous bacteria, as in infectious catarrhs of mucous membranes, where the leucocytes, by migrating to the surface, remove the bacteria from the affected part. On the other hand, it has been proved that the power of killing bacteria does not rest with the leucocytes alone, but is possessed in a much higher degree by the blood serum, lymph, and tissue fluids generally, and also by the fixed tissue cells which may act as phagocytes. Further, the leucocytes ingest solid particles of all kinds seemingly as readily as germs. They can be made to ingest nutritive particles, fat globules, pigment granules, red blood corpuscles, etc., by mere physical contact therewith, or after stimulation with various chemical solutions. Therefore the suggestion that phagocytosis is fundamentally of a nutritive nature seems as reasonable as that it is protective. In many cases, indeed, it can be shown to have a hurtful and not a beneficial action, as when leucocytes containing virulent bacteria migrate to healthy parts and there form a new focus of disease. The mere imprisoning of the bacteria within the protoplasm of the leucocyte does not render them harmless, as they may not only preserve their virulence intact, but increase it, since the leucocyte protoplasm may form a suitable and favourable bacterial nidus. Phagocytosis, while often playing an important part, cannot be held to be an essential phenomenon of inflammation, as it may be entirely absent in typical instances thereof. The leucocyte emigration is a wider phenomenon. It leads to an accumulation of leucocytes in the inflamed area which may be conveniently spoken of as a leucocytosis. The amount of leucocytosis varies within wide limits, being dependent upon several factors, the chief of which is the nature and intensity of the noxa and the anatomical structure of the affected part. While emigration from the blood-vessels is the chief source of the leucocytes, some, never many, come in certain cases from the wandering cells of the tissues, and others from an actual division of leucocytes *in situ*. They are rarely if ever produced by the transformation of fixed tissue cells, although this has been said to occur by more than one observer through the degeneration of muscle cells. According to the more restricted conception of inflammation, the presence of blood-vessels, either in an area or near it, is necessary for the possibility of the production of inflammation, and inasmuch as leucocyte emigration is the chief source of the leucocytosis which constantly occurs in its establishment, it must be regarded as being one of the essential phenomenon of such inflammatory processes. According to the wider acceptation of inflammation, which holds that blood-vessels are not at all necessary, the leucocytosis becomes still more important. The cornea is the structure which has been most widely chosen for experimentation in this relationship, for it is anatomically a non-vascular tissue. Its marginal parts (sclerotic) are so plentifully supplied with blood-vessels, that it is hardly possible to set up irritation in its central parts without transmission to these

peripheral vessels. Therefore it is practically a vascular tissue. In the invertebrate kingdom, on the other hand, the tissues may all be regarded as non-vascular, as there is no closed system of blood-vessels. They have, accordingly, been selected by many workers as affording a wide field for experimentation and investigation. Without entering into particulars, it may be stated generally that a more or less marked leucocytosis always appears in the irritated area. In many of the lower forms it seems to be the dominant factor, many of the leucocytes afterwards assisting in the process of repair. In the higher forms the leucocytosis is still very prominent, but the process of repair seems to be relegated entirely to the fixed tissue cells. Leucocytosis, therefore, appears to be an invariable and essential phenomenon of inflammation, whether the wide or restricted conception of the term be adopted. What purpose does it serve? We have already seen that many of the leucocytes act as phagocytes, and that it is at least probable that phagocytosis has a nutritive rather than a protective value. It is possible that this function is possessed by all the leucocytes, though other functions are also possible. Many of the leucocytes degenerate and break down, producing substances which dissolve in the tissue fluids and prove hurtful to bacterial life and activity. They may, in other words, through degeneration or otherwise be largely instrumental in producing substances which confer bactericidal properties upon the body fluids. Others may produce fibrin ferment and lead to the production of fibrin threads or masses within the inflamed area. Further, when recovery is taking place, solid masses of fibrin must be dissolved previous to their absorption, and this must be done largely through the agency of cells which may be leucocytes.

There is still another possible destiny of leucocytes which must be carefully considered, viz. the transformation of leucocytes into fixed tissue cells. There is no evidence whatever that this occurs in the case of the polymorphonuclear leucocytes, but with the mono-nuclear forms it is not so clear. These may leave the blood-vessels in considerable numbers, and collect in the affected area in the more chronic forms of inflammation, in which we subsequently get a marked fibrous tissue formation or fibrous hyperplasia. It has not yet been shown, however, that the mono-nuclear leucocytes contribute in any way to this formation. Histological methods have been so improved of late that it is possible to differentiate clearly the various cells of a tissue in the many changes which they may undergo, and so far no absolute proof of the conversion of mono-nuclear leucocytes into connective-tissue corpuscles has been brought forward. Metschnikoff and others have claimed that this does occur in the lower tissues of animal life, as in the tadpole's tail, and although it has never been shown to exist in the higher forms, yet it must be held to be possible, as it has never been definitely excluded. 2. Escape of the Red Blood Corpuscles.—This is a purely passive process dependent primarily upon the change in the vessel walls, and hence is usually well marked in severe inflammations. The nature of the irritant has also considerable influence, some irritants causing it much more readily than others. The greater density of the blood owing to the escape of the exudate, the adhesiveness of the lining membrane of the vessel walls, and the blood pressure, may all assist the process. The escape of the red blood cells from the blood-vessels is known as diapedesis. It subserves no special function. 3. Escape of the Blood Plates.—This takes place about the same time as the escape of the other corpuscular elements. It is somewhat difficult to follow, and comparatively few observations have been recorded, but so far as we know it has no significance in inflammation.

V. *The Rôle of the fixed Tissue Cells.*—The fixed tissue cells play an important part in all inflammations. The changes which they undergo depend mainly upon the nature and degree of intensity of the irritant. They may be divided into two stages, a degenerative or retrogressive stage and a regenerative or progressive one. 1. The Degenerative Stage.—This varies within wide limits, sometimes showing swelling and granulation of the protoplasm, and sometimes fatty, mucoid, vitreous, or other obvious degeneration. At other times the changes are so slight that they cannot be detected histologically, but they are always sufficient to cause disturbance of function. The matrix also undergoes degenerative changes. It loses its elasticity, and fails to exert the normal pressure upon the walls of the blood-vessels. Its resisting power is diminished, and it may show the same obvious degenerations as the cells, or even be dissociated and liquefied. Hyaline degeneration is common in chronic forms of inflammation. The direct action of the noxa is the main cause of these changes, but the absorption of exudate by the cells and tissues may greatly assist it. The exudate is often rich in mucin, which causes swelling, softening, and mucoid degeneration of the tissues. The fixed tissue cells also become detached. In the case of the connective-tissue cells they separate from the fibrous bundles, and in the case of gland tubes from their attachment to the basement membrane or wall of the tubes. The liberated cells may break up and disappear, or they may show signs of division and ultimately lead to multiplication. 2. The Regenerative Stage.—The newly-formed cells may produce new tissue after their own kind, and thus bring about repair of lost tissue. This can be well studied in the cornea. If its central part be injured, *e.g.* by a solution of zinc chloride, a loss of tissue arises at the injured point. The blood-

vessels at the margin of the cornea dilate, etc., and give rise to an exudate. Leucocyte emigration and the other typical signs of inflammation are duly set up. After a time changes are seen in the corneal corpuscles surrounding the injured area. They are seen to swell and proliferate, producing new cells. Some of them attain a considerable size, producing large cell masses each with many nuclei. The exudate and leucocytosis subside gradually, the peripheral blood-vessels return to their normal condition, and the newly-formed corneal corpuscles proceed to form new fibres, and in a short time all signs of the corneal injury have disappeared. This rapid and perfect healing is usually well exemplified in corneal ulcers. Much the same changes occur in vascular tissues, but the blood-vessels generally take a more active part, especially in considerable inflammations. The lining endothelial cells are about the first structures to be affected by the noxa, and it is only after they have been affected that escape of exudate and leucocyte emigration become possible. Later they may, like the connective-tissue cells outside, become detached and proliferated. The exact time at which the degenerative stage ends and the regenerative begins does not permit of delimitation. A certain time must elapse before regeneration can commence (five to twenty-four hours or more), and it is rarely of any extent until after the third day, the time required being largely dependent upon the character and intensity of the irritant. In the same way it may be indeterminate in its ending, and result in various forms of hyperplasia. This is especially seen in chronic inflammations. The degenerative stage may be very slightly marked, while the regenerative is very pronounced. The interstitial fibrous tissue which is so abundantly formed in cirrhosis of the liver and kidney is a good instance in point. The new formation of fibrous tissue is here looked upon as inflammatory, since it is due to the continued action of some irritant. On the other hand, somewhat similar changes may arise entirely independently of inflammation. In granular contracted kidney there is primarily a degeneration and atrophy of the renal epithelium, ending in its destruction over certain areas, and keeping step therewith there is a compensatory overgrowth of the fibrous interstitial tissue. Inflammation has nothing to do with these changes, although they give a renal cirrhosis practically indistinguishable in its later stages from that produced by inflammation. In the same way the regenerative processes of inflammation must be carefully distinguished from those of repair. They are not synonymous, though it is by no means easy to distinguish between them when they are present together, or when the one follows at once upon the other. This does not necessitate the exclusion of the healing processes from inflammation. When the blood no longer carries injurious substances, or when the influence of the irritant is at an end, normal blood will circulate through the affected vessels, conveying healthy nourishment to their walls and the tissues around, and thus gradually restore them to their healthy state (the *vis medicatrix naturæ*). The exudate is absorbed, the leucocytosis disappears, and in slight cases the tissues regain their normal structure and functions, but in more severe cases there is an actual loss of tissue which must be restored. There is a general tendency for lost tissue to be replaced by tissue after its own kind. When the new tissue is exactly like that which was lost the healing is perfect, but this rarely occurs in the highly specialised tissues. Bone and ganglion cells do not proliferate at all, and cartilage cells only very slightly. Striped muscle cells have considerable powers of proliferation, except in the case of the heart, where there is none. Non-striped muscle cells proliferate comparatively freely, and so do neuroglia cells and glandular epithelial cells, while covering epithelial cells, ordinary connective-tissue cells, and endothelial cells, have great and rapid proliferative powers. Many losses of tissue are consequently replaced by an inferior form of tissue. In the heart, for example, a loss of substance is replaced by fibrous tissue. This occurs also largely throughout the internal organs. Connective tissues in the main replace lost structures in the central nervous system (through proliferation of the cells of the vascular sheaths and neuroglia cells), liver, and kidney. Gland cells may be reproduced in slight losses in glandular organs, but they are usually of a low functional value. In more severe losses the gap is made good by fibrous tissue. The remaining healthy gland tissue has great power of compensatory hypertrophy. Nerve trunks, on the other hand, are largely regenerated. If a part of a peripheral nerve be destroyed, fibrous tissue first proliferates and fills the gap, and then the axis cylinders of the central end send out prolongations which perforate the fibrous tissues, and seeking out the distal end of the nerve in time re-establish connection with the peripheral nerve end organs. They acquire new medullary sheaths. In striped muscle fibrous tissue first fills the gap, and later new muscle fibres are formed which penetrate the fibrous scar more or less extensively, but rarely succeed in completely restoring the muscular continuity. Unstriped muscle fibres, on the contrary, are rarely re-formed to any extent, though their power of proliferation is considerable. New bone is often extensively produced, but always from the periosteum, endosteum, or perichondrium. Covering epithelium, whether squamous, mucous, or serous, is generally widely restored, and always from proliferation of the pre-existing epithelium, never by any other tissue. The subepithelial fibrous tissues are

also extensively and similarly restored by like tissues. The blood-vessels enter into the process of regeneration wherever there has been loss of tissue. New blood-vessels are formed and the new tissue appears around them. This may be easily followed in the healing of an ulcer. Its floor soon becomes covered with little red points which are known as granulations. Each consists of a vascular loop surrounded by cells of various types which may be arranged in three groups: (1) Small round cells with polylobular, fragmented, or single deeply staining nucleus, and with or without oxyphil granules. (2) Large uninucleated cells with a clear protoplasm. (3) Cells of varying size (mostly larger) and shape with a bright oval nucleus and abundant protoplasm. Their relative proportions vary greatly. The last tends to be deeper than the first two, which are probably both leucocytes. The regeneration of the fibrous tissue is apparently performed entirely by the third type, which is indubitably a connective-tissue cell. It assumes many different shapes, such as spindle, star-shaped, irregular, etc. It is often called an "epitheloid cell." It is seen to secrete fibres either at its side or end. Large cells with many nuclei (giant cells) sometimes appear in granulation tissue. They are epitheloid cells which have become altered by some special character possessed by the noxa, *e.g.* the tubercle bacillus, which can inhibit the division of the protoplasm, but not that of the nucleus. Similar cells are often seen around foreign bodies such as silk ligatures. The new blood-vessels of granulation tissue always arise as buds from the pre-existing capillaries of the inflamed area. They are at first solid (usually) outgrowths which come to bend round and join other similar outgrowths or pre-existing capillaries. They become vacuolated and hollow, and nuclei appear in their walls, which later become segmented into endothelial plates like normal capillaries. The blood soon makes its way through these new vessels from the mother capillaries. The gap of the ulcer will thus in time become filled up by this newly-formed vascular fibrous tissue, and its surface at the same time gradually covered by epithelium. The time which this takes will vary greatly according to circumstances. The most important of all the disturbing factors is the presence or continuance of infection. The amount of inflammatory exudate may be taken as a good practical guide to the probable duration of the inflammation, especially of its later formative phases. The exudate, wherever it is not absorbed by the lymph or blood, must be replaced by granulation tissue, a much slower process. Extensive exudates are followed by prolonged granulation tissue formation, which may lead to extensive fibrous thickenings and adhesions. These conditions hold good for deep (*e.g.* in muscles, bones, brain) as well as for superficial inflammations. Considerable transformation may afterwards take place in the newly-formed tissues The capillaries may largely disappear, and the fibrous tissues increase in number and density. Elastic fibres may appear and the connective-tissue cells greatly diminish in numbers. The ultimate cicatricial tissue is thus often denser, less elastic, less vascular, and more vulnerable than the original tissue which it has replaced. This ultimate development is often slow, and may not be reached for months.

The fixed tissue cells of an inflamed area usually exhibit other changes not referred to above. They may be seen at almost any stage of inflammation to have engulfed leucocytes, bacteria, pigment particles, remains of red blood cells, necrotic tissues, etc. They may do so while still *in situ* or after becoming free. They may show a considerable range of movement. It is probable that they possess and exert a certain degree of selection, for they may be brought in contact with bacteria without at first ingesting them, though they may do so later. This has been explained by the suggestion that the bacteria first exert a negative and later a positive chemotaxis, particularly when inert or degenerated. The phenomenon of phagocytosis is thus exhibited both by leucocytes (microphages) and by fixed tissue cells (macrophages), and if it be of a nutritive value in the former, it is probably so also in the latter.

It is obvious that all the changes which the fixed tissue cells undergo in inflammation cannot subserve a useful purpose, though some of them do. The earlier degenerative changes cannot be beneficial, but many of the later regenerative processes may be. The latter are, however, not always present, or they may be so slight as to have no practical influence. Again, they may be excessive and altogether beyond the requirements of the tissue, and in such cases they may do obvious harm. It is not the necessities of the tissues which call them forth, but the nature and the intensity of the noxa reacting through the vital qualities of the tissues themselves. They are also influenced by the nature and conditions of the structures affected.

Nomenclature used in Inflammation.—(i.) *Terms signifying Inflammation.*—It is customary to refer to inflammation of an organ under a term made up of the Greek (usually) name for the organ with the termination -itis appended. Hence we say peritonitis, pleuritis, endocarditis, pericarditis, meningitis, bronchitis, etc. The prefixes peri and para attached to these terms indicate that the serous covering or tissues around the organ are inflamed, as perihepatitis, perisplenitis, parametritis, etc.

(ii.) *Duration of Inflammation.*—The course of an inflammation is sometimes short, sometimes prolonged, hence we call it acute, subacute, or chronic.

(iii.) *Kinds of Inflammation.*—The factors

which modify inflammation are so many and their interaction so complex, that a satisfactory classification cannot be given. Various groupings are, however, in use, such as those depending on the site, the quantity and quality of the exudate, and the reaction of the tissues themselves.

1. *The Site.*—Those affecting the surface of an organ or tissue, *e.g.* the skin and mucous membranes, may be distinguished as superficial, while those affecting deep tissues, *e.g.* the interior of the brain, bones, etc., are called deep-seated. Inflamed tissues near a free surface, *e.g.* skin or mucous membrane, generally show the degenerative changes chiefly in the superficial cell layers, and also an abundant serous exudation (relatively more than into an abscess) which comes to the surface and escapes. It is mixed with cells (epithelial and leucocytes). In glandular organs the inflammation may affect the secreting structures, when it is called parenchymatous, or the supporting connective tissues, when it is called interstitial. Inflammations limited entirely to one or other of these structures are uncommon, but combinations occur frequently. Inflammations of muscular tissue and of the nervous system are similarly distinguished as parenchymatous and interstitial.

2. *The Qualities of the Exudate.*—(*a*) The inflammation is called serous when the exudate is a clear or slightly morbid fluid. It is most often seen in comparatively slight inflammations. (*b*) It is called purulent when the exudate is turbid or whitish by the plentiful admixture of leucocytes. When it occurs naturally in the human subject it may always be looked upon as caused by bacteria, though artificially, other agents such as turpentine, petroleum, and croton oil may produce it. Suppurations caused by chemicals are not so grave as those caused by bacteria, as they heal more rapidly and do not produce blood poisonings or metastases. (*c*) It is called fibrinous when the exudate undergoes a deposition of fibrin, either as a network, or homogenous, or flaky, or irregular, or granular masses, or as stratified layers. In the lungs it often forms a network, filling the alveoli, and is often spoken of as a croupous inflammation, a term which was originally applied only to the fibrinous deposit, or false, or croupous membrane, which forms upon the pharynx and larynx in certain severe inflammations, particularly in diphtheria. The term croupous was first of all used to describe the peculiar crowing noise of croup, and thence it came to be used to describe the false membrane which caused it. Its extension to other fibrinous inflammations, as in the lung, has nothing to support it, and should be discontinued. In deep-seated inflammations threads of fibrin may be occasionally seen, but its formation is always slight. Large deposits of fibrin are characteristic of severe inflammations of serous surfaces, such as the pleura or pericardium. (*d*) It is called hæmorrhagic when the exudate is mixed with blood, either from a plentiful diapedesis or from an actual rupture of blood-vessels. It sometimes indicates a grave form of inflammation, while at other times it points to some peculiarity of the irritant or of the tissues involved.

3. *The Reaction of the Tissues.*—The degenerative, liquefactive, and necrotic, etc., changes which the tissues show, and which are so largely dependent upon the nature and degree of intensity of the noxa, may also be used to describe special forms of inflammation.

This article does not attempt to give more than an explanation of what is meant when the term inflammation is used, or more than a short account of the various processes involved in its establishment, so as to furnish the busy student and practitioner with a workable and practical view of the subject, unburdened with a discussion of the many problems of a more strictly scientific interest with which its every aspect fairly bristles.

Inflation of Lungs.—*See* Asphyxia (*Artificial Respiration*).

Inflation of Tympanum. — *See* Ear, Examination of (*Inflation through Eustachian Tube*).

Influenza.

See also Angina Pectoris (*Etiology, Influenza*); Brain, Inflammations (*Acute Encephalitis, Etiology*); Bronchi, Bronchitis (*Acute Bronchitis, Etiology*); Dengue (*Diagnosis*); Epidemiology (*Seasonal Fluctuations*); Expectoration (*Influenza Bacillus*); Insanity, Etiology of (*Microbic Toxic Action*); Insanity, Nature and Symptoms (*Index of Etiological Varieties*); Leucocytosis (*Leucopenia in Influenza*); Lung, Tuberculosis of (*Predisposing Factors, Influenza*); Malaria (*Diagnosis*); Meninges of the Cerebrum (*Acute Simple Meningitis, Etiology*); Nerves, Multiple Peripheral Neuritis (*General Etiology*); Nose, Examination of (*Character of Secretion, Bacteria in Influenza Rhinitis*); Nose, Post-Nasal Adenoid Growths (*Etiology*); Nose, Post-Nasal Adenoid Growths (*Operation for, Sequelæ*); Nose, Accessory Sinuses, Inflammation of (*Etiology*); Ophthalmoplegia

(*Etiology*); Pleura, Diseases of (*Acute Pleurisy, Etiology*); Pneumonia, Bacteriology of (*Introductory*); Pneumonia, Clinical (*Etiology, Influenza*); Post-mortem Methods (*Bacteriological Investigations, Influenza*); Puerperium, Pathology (*Fever, Diagnosis*); Relapsing Fever (*Diagnosis*); Thyroid Gland, Medical (*Acute Thyroiditis, Causation*); Typhoid Fever (*Diagnosis*); Typhus Fever (*Diagnosis*).

Definition

Influenza is an acute, specific, infective febrile affection, characterised by its remarkably sudden onset, after an incubation of about two or three days, the many symptomatic phases it presents, the singular predisposition to other secondary infections which it induces, its disastrous effect upon the heart, and the protracted convalescence which follows in its wake. In itself by no means a mortal disease, influenza exercises a far-reaching and malignant effect upon the death-rate, which surges far above the average during the few days or weeks of its epidemic or pandemic prevalence. Catarrhal symptoms, although often present, are not an essential feature of the disease. The victims of influenza are adults, who perish from pneumonia or bronchitis; and the aged, who sink from heart exhaustion. Children, while prone to the disease, enjoy a comparative immunity from its complications and dangers; in them a sharp febrile movement, with or without an evanescent rash of an erythematous nature, constitutes the main feature of the attack. Unlike the poison of typhus, the virus of influenza is not rendered inert by oxidation from contact with atmospheric air, but rather the contrary.

Bacteriology

The specific micro-organism of influenza was identified by Pfeiffer of Berlin, and independently by Canon of the same city in 1892. It is a very small aerobic, non-motile bacillus, which does not grow at temperatures below 28° C. (82·4° F.). Spore formation has not been observed. Its diameter is $0{\cdot}2\mu$ to $0{\cdot}3\mu$, and it is only about $0{\cdot}5\mu$ in length. It occurs singly or united in chains of two, three, or four elements. It is quickly destroyed by desiccation. The thermal death-point is 60° C. (140° F.) with five minutes' exposure. This bacillus was discovered by Pfeiffer in the grey mucus of the bronchial mucous membrane and in the purulent bronchial secretion; by Canon in the blood of patients suffering from epidemic influenza in 1892. Canon's observations as to the presence of the influenza bacillus in the blood lack confirmation. The deep staining of the ends of the bacillus with dilute carbol-fuchsin (Ziehl) solution or methylene blue (Löffler) solution, with heat, causes it to resemble a diplococcus (Sternberg). Pfeiffer's bacillus is present not only in the nasal and bronchial secretions, but also in the substance of the lung, where it may set up pneumonia in both adults and children. It may be localised in the spleen, pericardium, endocardium, middle ear, central nervous system, and pleura. In severe cases the bacillus is also found in the peri-bronchial and sub-pleural lymphatics (Goodhart).

Historical

It would be foreign to the object of this article to enter at any length into the history or bibliography of influenza. When preparing his classical memoir on the Mortality of Ireland, published in the Census of 1841, the late Sir William Wilde, M.D., learned many curious facts connected with this disease and its epidemic outbreaks in early times in Ireland. It is described in a medical manuscript of the fifteenth century under the names of *Fuachd* (*i.e.* cold or chilliness) and *Slaodan* (*i.e.* a cough or cold), and is mentioned in the *Annals of the Four Masters* as epidemic in Ireland in the fourteenth century. A disease, the symptoms of which answer to those of influenza, is also alluded to in certain early Gaelic manuscripts under the term *Creathan* (*i.e.* a shaking or trembling).

Finkler points out that the year 1510 is of especial importance in the history of influenza, as we here meet with the pestilence for the first time as a pandemic disease. It overran all Europe. Its historian, Dr. Thomas Short, thus describes the outbreak:—"The disease called *Coccoluche*, or Coccolucio (because the sick wore a cap or covering close all over their heads), came from the island *Melite* in *Africa*, into *Sicily*; so into *Spain* and *Italy*, from that over the *Alps* into *Portugal*, *Hungary*, and a great part of *Germany*, even to the *Baltic Sea*; every month shifting its situation with the wind from East to West, so into *France*, *Britain*, etc., *Valeriola*, *Pechlin*, etc. It attacked at once, and raged all over *Europe*, not missing a family and scarce a person. A grievous pain of the head, heaviness, difficulty of breathing, hoarseness, loss of strength and appetite, restlessness, watchings, from a terrible tearing cough. Presently succeeded a chillness, and so a violent cough, that many were in danger of suffocation. The first days it was without spitting; but about the seventh or eighth day much viscid phlegm was spit up. Others (though fewer) spit only water and froth. When they began to spit, cough and shortness of breath were easier. None died except some children. In some, it went off with a looseness; in others, by sweating. Bleeding and purging did hurt. . . . Where blood was let, the disease proved malignant and pestilential, being attended with a violent, cruel, and unheard-of malignity, and made bad work."

The reader will admit that the foregoing description is a faithful portraiture of the influenza of our own day.

From time to time during the succeeding centuries epidemics of the disease occurred. An outbreak in the spring of 1743 was well described by J. Huxham, who adopted the name "influenza" for the complaint in the sentence —"This fever seemed to have been exactly the same with that which, in the spring, was rife all over Europe, termed the 'influenza.'" It is noteworthy that the French name for the malady, "la grippe," came into use also in connection with the outbreak of 1743. Biermer writes: "In France the 'influenza' has generally been called la grippe since the epidemic of 1743. This designation is probably derived from *agripper* (to attack), and not likely from the Polish word *chrypka* (*raucedo*), as T. Frank believes." In September 1758 influenza became epidemic in Edinburgh and in Scotland at large. Dr. Robert White, F.R.S., Professor of Medicine in the University of Edinburgh, the historian of this outbreak, observes that early in September, before the influenza appeared, a disease existed among horses in Perthshire—"the horses were observed to be more than usually affected with a cold and a cough." This is the first mention of the coincidence of an epizootic with influenza. In 1775 again Dr. Thomas Glass of Exeter notes that in his part of the country, in September, "many horses and dogs were severely afflicted with colds and coughs." Dr. Fothergill, in his *Sketch of the Epidemic Disease which appeared in London towards the end of the year* 1775, says that "during this time horses and dogs were much affected—those especially that were well kept. The horses had severe coughs, were hot, forbore eating, and were long in recovering. Not many of them died that I heard of, but several dogs."

Coming down to our own time, the occurrence of the so-called "pink-eye" among horses has been frequently observed since the great pandemic outburst of influenza in the autumn and winter of 1889. In his work on *Horses and Stables*, Lieut.-General Sir F. Fitzwygram, Bart., says: "The term 'pink eye' has of late years been applied to a particular form of influenza manifested by symptoms of a peculiarly marked nature, notably that of a remarkably clear pink coloured condition of the conjunctival membrane accompanied by a swollen or œdematous state of the conjunctivæ." He adds that "pink eye" is now generally recognised as being merely a modification of the catarrhal form of influenza. It may be remarked that in "pink eye" there is a noticeable tendency towards the formation of fibrinous clots or thrombi in the cavities of the heart and in the larger arteries, especially in the pulmonary system—a condition which not infrequently leads to unexpected and even sudden death.

In his Report to the Local Government Board (England) on the great epidemic of 1889-90, Dr. H. F. Parsons points out that, prior to the outbreak of influenza in London and some other parts of England, and also in some continental countries, a disease termed "influenza" prevailed among horses. In certain large stables in London numerous horses were affected by this epizootic in October 1889, some six weeks before the human epidemic made its appearance. Dr. E. Symes Thompson, the author of *Influenza, or Epidemic Catarrhal Fever*, says that he was so strongly impressed with the intimate connection between "pink eye" in horses and human influenza, that in the early part of December 1889 he wrote to the *British Medical Journal*, calling attention to the prevalence of an equine epizootic, and suggesting that it would not improbably prove the forerunner of an outbreak in man. These facts appear to establish a relationship between human and equine influenza, but a closer examination throws considerable doubt on such a relationship. In the first place, human influenza is reproduced in animals by inoculation only with difficulty and most inconstantly. Bollinger of Munich also believes that equine influenza and human influenza are essentially distinct diseases, because in the great horse epizootic resembling influenza which overran North America in 1872-73, there was no corresponding epidemic among human beings. Again, in the outbreak of 1889-1890, persons having to do with horses were not observed to be specially or earliest affected, while influenza prevailed epidemically at certain places, such as Newmarket, where no similar disease among horses was affirmed. Lastly, the bacteriology of the two diseases differs. Pfeiffer's bacillus is wanting in horse influenza, while Schutz, in 1888, cultivated a streptococcus from the lymphatic glands of horses suffering from "epizootic influenza," known in Germany as *Druse des Pferdes*, which he believes to be the specific infectious agent in this disease, and to which the name of *Streptococcus coryzæ contagiosæ equorum* has been given.

After the epidemic of 1758 the chief outbreaks of influenza in the eighteenth century were in America in 1761, Europe in 1762, Asia and Europe in 1781-82—"one of the most widespread pandemics of this disease that have ever occurred," according to Finkler; Europe in 1788-89, and America in 1789.

As pandemics of influenza in the nineteenth century, Finkler quotes the outbreaks of 1802-1803, 1830-33, 1836-37, and 1847-48, as being generally recognised. Hirsch adds to this list the epidemics of 1850-51, 1855, 1857-58, and 1874-75. Lastly, comes the great pandemic of 1889-90, with its repeated "flarings" during the succeeding decade—flarings which have not yet ceased to appear.

The history of this notable outbreak begins in May 1889, when influenza appeared in Bokhara, in Central Asia, thence travelling north-

eastwards to Tomsk in Siberia, north-westwards to St. Petersburg, and westwards to the Caucasus and Southern Russia. In the last quarter of the year named the epidemic spread rapidly from Eastern Europe to the central and western countries. Early in 1890 it extended to America, Africa, and Australasia, "so that," says Finkler, "we may truthfully designate its appearance in 1889-90 as pandemic." "The high flood of the pandemic," observes the same writer, "flowed over the whole globe in the space of a few months."

As it travelled westward the disease was known by some name connecting it with the country whence it spread. Thus, in Siberia it was the "Chinese distemper"; in Russia, it became "Siberian fever"; in Western Europe it was the "Russian influenza."

Etiology

A theory was propounded in 1890 by the Hon. Rollo Russell that the pandemic of 1889-90 might possibly be traced to the diffusion of atmospheric dust in the wake of a great flood which had occurred early in 1888, in that part of China through which the Hoang Ho, or Yellow River, flows. This theory is everywhere discredited, although Dr. E. Symes Thompson in 1890 threw out the suggestion that dust in the air, whether Chinese dust or meteoric dust, might supply the "raft" for the aerial conveyance of the microbe of influenza. The presence and diffusion of dust in the air, according to Thompson, might be the condition which determined the prevalence of the disease, and might be adequate to convert a local epidemic into a universally distributed or pandemic wave. In the *British Medical Journal* of May 9, 1891, the present writer offered a meteorological explanation of the transference of influenza from point to point on the earth's surface.

Influenza had been raging in North China since the beginning of the winter of 1890-91. It appeared in several towns in the United States of America early in March 1891, and spread eastwards from Chicago, Pittsburg, Cleveland, and several other places in Ohio and in Iowa, finally invading New York and other cities near the eastern sea-board. About the second week in April an outbreak occurred in Norway, 891 cases being reported in one week in Christiania, and also in Sweden, notably in Göteborg. Almost simultaneously a severe outbreak took place in Yorkshire and Lincolnshire, extending thence southwards and westwards to the Midlands and the south-eastern counties of England.

The presumption is that the influenza microbes were wafted from China across the Pacific to North America by the air-currents connected with a winter barometric depression lying over the North Pacific. Subsequently the germs would be distributed along the eastern sea-board of the North American continent by the north-westerly winds of the American winter anticyclone or area of high atmospheric pressure, which results from the piling up of masses of cold, dense air over the land in winter.

Proceeding on the lines of the same hypothesis, the agency by which the influenza outbreak in North-Western Europe was produced was a spring anticyclone over Scandinavia and that part of the North Atlantic to which the name of the "Norwegian Sea" has been given. Reference to the weather charts published in the *Daily Weather Report* of the Meteorological Office, London, will show that almost uninterruptedly from the 1st to the 25th of April 1891 an anticyclone hung over either Scandinavia or the Norwegian Sea, or over both these districts. Under such circumstances, easterly and north-easterly winds — descending from the higher strata of the air, and hence so keen and dry as these winds are known to be—played upon the very districts in Sweden and Norway, and England, which were simultaneously affected with epidemic influenza.

In his Report to the Local Government Board for England and Wales on the *Epidemic Influenza of* 1889-90, Dr. H. Franklin Parsons observes that the progress of the epidemic in a direction from east to west, *i.e.* in a direction contrary to the winds prevailing at the surface of the ground, is at first sight opposed to the hypothesis of an infective material being carried by the air. Dr. Alexander Buchan, LL.D., Fellow of the Scottish Meteorological Society, explains this progress by supposing the material to be caught up by the ascending currents into the upper regions of the air, where currents prevail in different directions from those at the surface of the earth, and brought down again in other places where descending or anticyclonic currents exist. The late Rev. Clement Ley, Inspector of the Meteorological Office, London, however, informed Dr. Parsons that the atmospheric conditions prevailing during winter over the central and northern regions of the continents of Asia and Europe are such that upward currents cannot occur there at that season—the cold which prevails over the great continental tract in question renders it throughout the winter an area of high barometric pressure and descending air-currents. This reclamation, nevertheless, does not affect the hypothesis given above as to the aerial transference in the winter of 1890-91 of influenza germs from China to North America, and thence to Scandinavia and the British Isles.

The mode of spreading and the prevalence of influenza are apparently quite independent of season, and weather, and climate — a circumstance which distinguishes influenza from epidemic bronchial catarrh. Petrus Salius Diversus wrote — "Et tempore frigidiori et calidiori, et flante tam Austro quam Borea et

pluvioso et sereno cœlo, peragravit hasce omnes Europæ regiones, et omnia loca indiscriminatim."

Nor do overcrowding, want of ventilation, and other sanitary shortcomings seem to favour the spread of this disease. In the epidemic of 1889-90, as observed in Dublin, influenza spread as early and as quickly through the suburbs as it did through the crowded streets and alleys of the city. This is in marked contrast to the behaviour of such infective diseases as typhus, small-pox, scarlatina, and measles.

Several writers advance the view that influenza is not so much a miasmatic as a miasmatic-contagious disease. Thus, Dr. P. Duflocq, chef de clinique in the Faculty of Medicine in Paris, concludes an elaborate article on the clinical varieties of influenza observed in that city in December 1889 and January 1890, with these words:—"La grippe semble donc être une maladie à la fois épidémique et contagieuse, et la période d'incubation serait de deux jours." According to this view the influenza germs produce their toxic effect chiefly by direct transmission, but also through the air outside of the human body.

A third school regards the disease as essentially and exclusively contagious, that is, transmissible from the sick to the healthy just as typhus, or scarlatina, or measles, or small-pox is. According to this doctrine influenza spreads from person to person directly or indirectly. The most usual mode of infection is by direct contact with those ill of influenza. But there is some evidence to show that healthy persons who have been in attendance on influenza patients may carry the infection to others while remaining well themselves. And, again, instances are recorded in which the disease has been spread through the agency of inanimate objects such as baggage, furniture, and even food-stuffs. Bäumler, for example, says that the first case of influenza in Basle occurred in a person who had been engaged in unpacking a bale of merchandise, which had recently arrived from the greatly infected Magazine du Louvre in Paris. Finkler, who quotes this observation, also narrates the following incident:—"An officer of the ship *La Bretagne*, which was anchored in December 1889 in the harbour at Brest, became ill at his home in Brest on 11th December, three days, it is said, after the arrival of a number of packages from Paris, which were first unpacked by the officer. These packages then carried the disease to his ship, while the vessels *Borda* and *Austerlitz*, which were anchored beside this vessel, are said to have escaped the infection."

So long as the miasmatic or pandemic view of the origin of influenza was entertained, there was no need to seek for a period of incubation, the virus being supposed to be already "hatched" at the time of its reception into the human system—that is, at the time of infection. It was indeed even then observed that in most cases there was an appreciable interval between the reception of the poison and the development of the symptoms, the most common duration of this interval being one to three days. We now know that this is a true period of incubation. In the last decade a vast amount of evidence has accumulated as to the communicability of the disease, and the latent or incubative period has been determined at two days on the average. Dr. Dawson Williams considers that the usual duration of this period is probably two or three days. This agrees with Dr. Parsons' estimate exactly. Sometimes the symptoms of an attack seem to develop almost simultaneously with exposure to infection. In one instance, observed by the writer, a lady visited a friend ill of influenza at 2 P.M. Three hours later she was attacked with symptoms of the disease—chills, weakness, coryza, lacrymation, stuffing of the nostrils, loss of smell and of taste. Dr. George Neale of Cadoxton, Glamorgan, in his own case, had a rigor an hour after visiting his first influenza patient. On the other hand, the period of incubation has been prolonged to several days, although a duration of latency exceeding seven days must be regarded as most exceptional.

In considering the effect of an epidemic of influenza upon the public health, we are, unfortunately, at a serious disadvantage in having no system of registration of disease in the United Kingdom. Experience of recent outbreaks, however, shows that, with the coming of influenza, the whole "epidemic constitution"—to use Thomas Sydenham's classic phrase—changes rapidly for the worse. The power of resisting disease is lessened among a people smitten with this plague, and extreme languor with prostration passes over the population like a destructive tidal wave.

The writer's observations on the epidemic of 1889-90 in Dublin extended over a period of nearly three months, from early in December 1889 up to the end of February 1890. The two earliest cases which came under his notice dated from Thursday and Friday, 5th and 6th December. One of these two patients consulted him on Monday, 9th December, having been ill from Thursday the 5th. He was a clergyman, aged fifty-two, in whose case the prominent symptoms were headache, tender eyes, palpitation, insomnia, and loss of strength. The outbreak was at its height in the first half of the following January. Towards the close of that month its epidemic prevalence waned quickly, but in the middle of February a recrudescence occurred. The following conclusions are justified by the facts:—

1. The epidemic of influenza was more pernicious to the population of Dublin than the extreme cold of January 1881.

2. It slew its victims, not so much directly,

as by means of complications and sequelæ, affecting the breathing organs and the heart.

3. It spared the lives of children of tender years, but killed large numbers of adults and those advanced in life.

4. Its effect upon the death-rate was sudden and pronounced, and lasted for at least seven weeks, or throughout the month of January and first half of February.

The virus or contagium of influenza, when once introduced into the body, acts primarily and quickly on the nervous system, producing the phenomena of an acute pyrexia, with singularly rapid pulse. In January 1890 a boy of seven and a half years had incessant nausea and vomiting for 24 hours, and profuse sweating, with a pulse as fast as in scarlet fever—140 per minute. He was one of a family of five children, all of whom had influenza together, their ages ranging from thirteen to four years. The mildest cases were those of the eldest and youngest. There was a bronchial catarrh in two instances. Very young children seem to enjoy a certain immunity from the disease, or to have it in a mild form—an ephemeral fever, followed by profuse sweating, and after a few days a tendency to slight catarrh. Hilton Fagge says that influenza "is sometimes dangerous to very young children," but he adds in a footnote that "Sir George Baker writes of the epidemic in 1762, *Leviter plectebantur infantes et liberabantur facillime.*" This is altogether the writer's experience also. In 1847 Dr. Fleetwood Churchill communicated to the *Dublin Journal of Medical Science* observations he had made on the epidemic influenza observed in January and February of that year among children in Dublin. In upwards of sixty cases, embracing children of all ages, from two months old to twelve or fourteen years, he had not a single death; so that he was led to conclude that, among children, although the epidemic was very general and severe, yet the danger was not very great when the disease was properly handled. This "child-type" of influenza presented itself to the writer's notice in 1890 in a large girls' school, of which he is one of the physicians.

Clinical Features

Adults in most instances suffer much more severely than children from the influenza poison. In them the attack as a rule comes on with lightning-like speed. A person apparently in perfect health is suddenly overcome by a feeling of general discomfort and profound depression. He feels chilly, or is shaken with a rigor worthy of an ague fit. His head aches. There are pains in his eyeballs, or "behind the eyes," and the eyeballs are exquisitely tender on pressure. Soon rheumatoid pains rack his body. They are particularly severe in the nape of the neck, small of the back, in the knees, and along the margins of the ribs. The patient is often sleepless, sometimes delirious. He may temporarily suffer loss of the special senses of smell, taste, and sometimes hearing. His eyes smart, tears overflow, he shrinks from the light (photophobia). Occasionally there is intense earache (otalgia). Strength ebbs away far more quickly than even in typhus, so that after a few hours he must take to his bed. Nor do the digestive and respiratory systems escape. The tongue becomes thickly coated with a creamy or blanket-like fur. There is a bad taste in the mouth, the breath is heavy or even fœtid. There is complete loss of appetite, amounting to a loathing against food. Nausea and perhaps vomiting add to the sufferer's distress. The bowels are constipated, but occasionally diarrhœa sets in as in an attack of cholerine. In another group of cases the brunt of the poison appears to fall on the respiratory tract. Cough, expectoration, a sense of oppression on the chest, a tightness in the breathing being common symptoms. In a word influenza seems to have the property of picking out the weak point in a person's constitution. If the patient is neurotic, nervous and neuralgic symptoms are likely to develop. Any old tendency to catarrh of either the respiratory or the digestive mucous membrane is at once intensified in the presence or in the wake of this strange malady. At the same time catarrhal symptoms may be badly marked or entirely absent. Dr. Parsons, in his Report on Epidemic Influenza in 1889-90, states that the comparative absence of catarrh led some observers to doubt whether that visitation had been one of true influenza. But a similar infrequency of catarrh had been observed on previous occasions. Dr. Parsons quotes the following authorities. Sir William Gairdner, writing of an outbreak which occurred in 1862, says: "But although catarrh is frequent and may be severe, the disease is essentially a fever and not a catarrh. Nay, the catarrh may be absent or insignificant; not unfrequently it is so." Sir Samuel Wilks also, writing of the epidemic of 1847, says: "Although a synonym for influenza is 'epidemic catarrh,' the latter was by no means a constant symptom; many of the worst cases, and especially the fatal ones, having no catarrhal symptoms whatever." In a report on the epidemic of 1782, the Royal College of Physicians of London state that "the symptom which universally prevailed, and which appeared to be almost a pathognomonic of the disease, was a distressing pain and sense of constriction in the forehead, temples, and sometimes in the whole face, accompanied with a sense of soreness about the cheek-bones under the muscles. This, now and then, was felt previously to the catarrh, and not unfrequently was followed by very little or no catarrhous affection."

Types

The protean forms which influenza presents

from a clinical aspect arise from the peculiar aptitude its poison has for fastening on the points of least resistance in the constitution. Of the many types described, perhaps the four following stand out most prominently:—

1. The neurotic, neuralgic, or rheumatic type.
2. The cardio-pulmonary type.
3. The gastric or gastro-intestinal type.
4. The febrile type.

It is proposed to give a brief clinical sketch of four cases, each illustrating a type observed in the epidemic of 1889-90 in Dublin.

I. *The Neurotic Type.*—One of the earliest cases which the writer saw in the epidemic of 1889-90 was that of a lady, who was seized on the evening of Friday, December 20th, 1889, and who was visited next morning by Dr. James Craig, one of the physicians to the Meath Hospital, Dublin. The following is this lady's own account of her attack:—"Friday, 20th December 1889, I went to the oratorio at St. Patrick's Cathedral apparently in my usual health. Shortly after entering the Cathedral I felt chilled, as if cold water was being poured down my back and legs. When I returned home I warmed myself at a good fire, was given some hot wine and water, and went to bed; then my face and head got very hot and uncomfortable, and pains began in my arms, shoulders, and legs. All night the pains were very bad, sometimes so sharp across the back of my chest that I could have cried out; and, although I felt burning to the touch, the cold-water sensation continued. I got no sleep that night. Next day, about twelve o'clock (mid-day), I was given a powder (salicylate of sodium), and in two hours afterwards another, which put me into a perspiration. The pains in my limbs got better, but my head began to ache badly, and all day I felt very ill. I suffered from great thirst. Saturday night slept better. Sunday morning about 5 A.M. I wished for a cup of tea, but could not taste it. I might have been drinking hot water. Sunday evening pains had quite gone. I had no headache. I got up for a while, but felt very weak. For several days I had no energy for anything, the least exertion tired me. My sense of taste did not return for four or five days. I also got a cough which was very troublesome. Temperature—Friday night 101°; Saturday morning 100°, evening 98·8°."

II. *The Cardio-pulmonary Type.*—On Monday, December 30th, 1889, Mrs. W., a lady, aged fifty-four years, somewhat frail and delicate, while out walking was seized with shivering and violent headache, and intense pain in the back and "bones." On reaching home she at once went to bed, feeling very ill and prostrate. Next day the tongue was thickly furred and dry. Her pulse was 132; respirations, 28; temperature, 103·3°. Having regard to the sudden onset of the illness and the symptoms, the attack was declared to be one of influenza.

On New Year's Day (third day) the pulse was 110; respirations, 28; temperature, 102·0°.

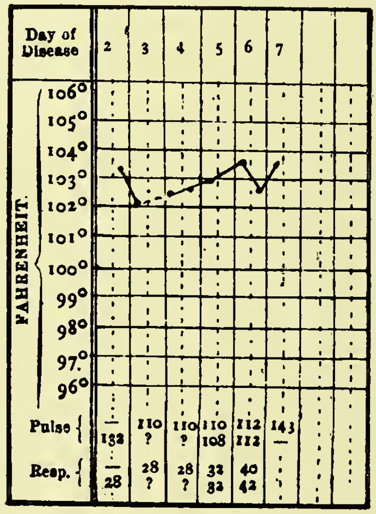


Chart I.—Mrs. W., aged 54.

The tongue thickly coated; eyes tender and lacrymation; complete anorexia; great prostration.

January 2, 1890 (fourth day).—The report was that she had a better night. Herpes was showing round the nostrils. Pulse, 96-100; respirations, 28; temperature, 102·4°. Severe stabbing or catching pain was complained of at the lower part of the left side of the chest. No physical signs could be detected, and a hot poultice relieved the pain.

January 3 (fifth day).—Pulse, 110; respirations, 32; temperature, 102·7°. A lymphy crepitation was now audible over the upper part of the left side of the chest, and on deep inspiration a fine pneumonic crepitation could be heard.

January 4 (sixth day).—Pulse, 110; respirations, 40; temperature, 103·2°. Dulness now existed, which was rapidly extending all over the left apex, where also a marked *frottement* could easily be recognised. At 6 P.M. Dr. Watson Pike, R.A.M.C., saw the patient in consultation and agreed in the diagnosis of influenza complicated with a left pleuro-pneumonia. Pulse, 112; respirations, 42; temperature, 102·7°. There was not a trace of expectoration, and scarcely any cough occurred. We considered the patient to be in danger, and continued the treatment, which consisted in free stimulation, frequent feeding, and quinine.

At 1.30 A.M. of Sunday, January 5th, the patient was sinking fast. Her pulse was failing, and the temperature was 103·3°. She rallied for a time, but at 6 A.M. another attack of cardiac failure came on. From this also she rallied, but at 10 A.M. she suddenly died.

As bearing on the diagnosis of this case, it is to be noticed that four—if not five—of the other members of this lady's family suffered from influenza either immediately before or after her illness.

Appended is the clinical chart.

The foregoing case illustrates the fact that influenza, while infrequently directly fatal, causes an indirect loss of life which is appalling, chiefly through complications affecting the respiratory, and, in advanced life, the circulatory systems. It has been said that influenza, while relatively less fatal, is absolutely more fatal than cholera.

The writer has seen fatal cases of influenzal bronchitis, pneumonia, pleuritis, and heart failure. The pneumonia, while producing the ordinary physical signs of acute croupous pneumonia, is often latent in its course, or accompanied by a profuse muco-purulent expectoration, with scarcely any rusty sputa. The ebbing of the strength in some of these cases in elderly people is something awful—it is often absolutely beyond control.

Younger subjects have a better chance of "pulling through." For example, a young lady, aged nineteen, was suddenly attacked with shivering, headache, and high temperature, with very rapid pulse, on Friday, December 6th, 1889. The fever lasted a few days, and then gave way, to be followed by a second rigor on Friday, December 20th, and urgent chest symptoms. On visiting her with Dr. Usher of Dundrum on the 22nd, her pulse was 124, respirations were 36, and the temperature 102·7° at 3 P.M. The left base was solid. Great dyspnœa, tubular breathing, and marked bronchophony were present. She was bringing up an abundant muco-purulent sputum—like that of bronchiolitis rather than acute lobar pneumonia. It was examined by Dr. Bewley, who reported that no tubercle-bacilli were present, but that various putrefactive microbes were found. On December 26th, her pulse was 120, respirations had risen to 48, and the temperature at noon was 101·6°. Dr. James Little saw her with Dr. Usher and me next day, when we found a new patch of pneumonic consolidation in the middle of the right back, and there were a few rusty-tinged sputa. From this time she steadily recovered, and went to Bournemouth with her father on January 23rd, 1890, "pretty nearly all right," as he expressed it in a letter to me.

III. *The Gastric Type.* — On Wednesday, January 8th, 1890, Mr. W. B. S. enjoyed a day's shooting in the County Wicklow. The following day he returned to town in his usual good health; but in the afternoon felt chilly, complained of headache and nausea, and felt utterly miserable. He went to bed early, but passed a wretched night—restless and sleepless. Next morning he complained of pains in the eyeballs, back of the head, and small of the back. Pulse, 84; temperature, 99·9°; tongue thickly coated; complete loss of appetite and nausea. He felt entirely prostrate, and expressed his belief that some fish which he had eaten for dinner had thoroughly disagreed with him. Two miserable days of sickness followed, the temperature rising on the morning of the 5th day to 103·1°. A short cough had set in, and the eyes were suffused and tender. There was constipation, and he complained of weight and fulness in the pit of the stomach. Dr. James Little saw him with me and thought it likely that the fever would run on for some time. A quiet day gave promise of a restful night, and this promise was abundantly fulfilled. He had an excellent

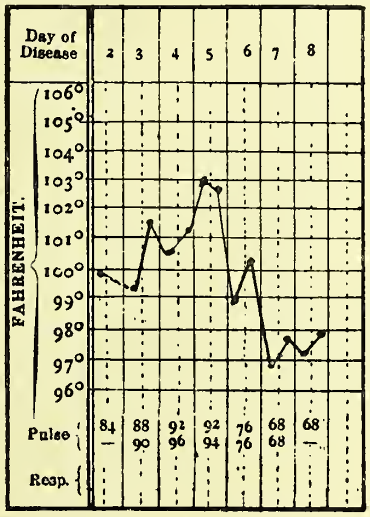


Chart II.—Mr. W. B. S.

night, partly due to 20 grains of phenazone, with 20 minims of tincture of gelsemium in a draught in four divided doses at bedtime. Next morning, pulse 76; temperature 98·7°, rising to 100·2° in the evening, but without any return of restlessness. Subnormal temperatures followed for a few days—96·4° being one observation. The tongue cleaned very slowly, and several days of extreme languor and weakness preceded final convalescence.

This gentleman's wife had, a few days previously, suffered from influenza, from which she was recovering when he fell ill. She nursed him and got a relapse, accompanied with cough, bronchial catarrh, and absolute loss of appetite. It is right to mention that Mr. S. had been subject to occasional "bilious attacks," as he described them. Except for these he had not known a day's sickness from his boyhood, and did not recollect when he had been obliged to keep his bed even for a day.

IV. *The Febrile Type.*—On New Year's Eve 1889 I received a note from a surgical colleague asking me to see him, as he had been taken ill the same afternoon while in his study. On visiting him in bed, he was still shivering at

times, and complaining of a distressing feeling of cold water streaming down his back. He stated that he had been out of sorts for three or four weeks, and it was quite evident that he

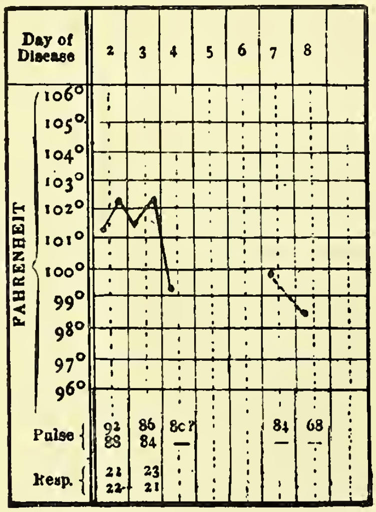


CHART III.—W. S.

had made up his mind that the attack was one of typhoid fever. His pulse was 92, the temperature about 100°. His tongue was very furred, and his eyes were injected with swollen lids. He had lost the senses of taste and smell, and complained much of rheumatoid or neuralgic pains in the back and limbs. Two restless feverish nights followed. So I asked our

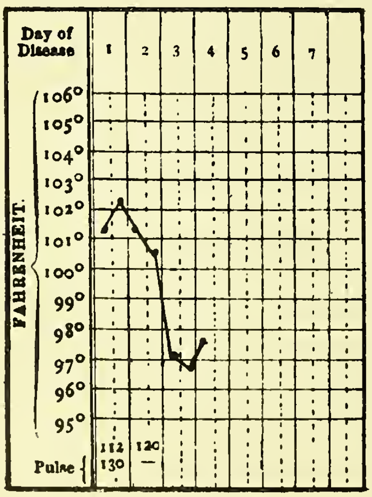


CHART IV.—Elsie M.

mutual friend, the late Dr. Samuel Gordon, to see him with me. At the end of the examination the patient said to Dr. Gordon, "Our friend Moore says I have influenza; do you think I have?" The answer was, "I do not think it, but I am sure of it."

There was in this case steady pyrexia for four days, as shown in Chart III., and then came profuse sweatings, lasting for several days. A slight elevation of temperature occurred on the evening of the seventh day, after which

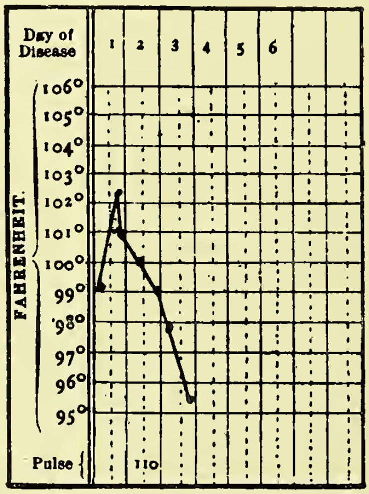


CHART V.—Dorothy O.

convalescence went on uninterruptedly. The weakness was for a time extreme.

This febrile type prevailed especially among children. The three charts, numbered IV., V., VI., show the febrile movement in girls, aged from 12 to 15. The marked subnormal temperatures on the third and following days are very noteworthy, and are so constantly present

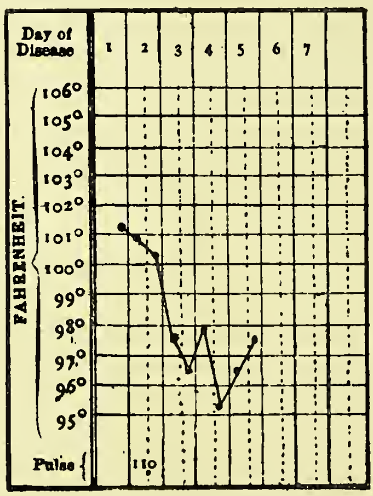
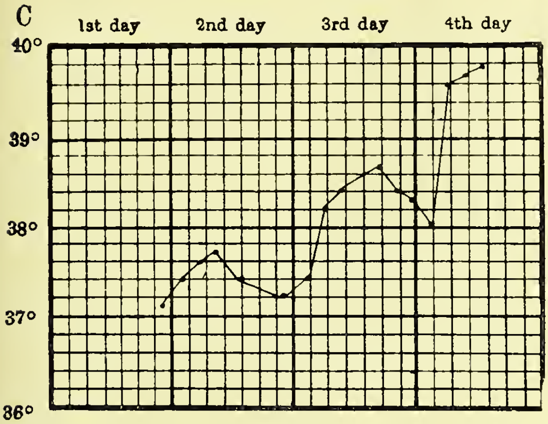


CHART VI.—Fanny C.

in the defervescing stage of influenza as to become an important diagnostic sign.

The febrile movement in even uncomplicated influenza is, as Wunderlich would say, "polytypical," or "atypical." This is shown by the charts already given, and, further, in a diagram containing three different types of temperature

range in influenza (which is reproduced), illustrative of a paper, "Zur Kenntnis des Fieberganges bei Influenza," by Dr. Otto Frentzel,

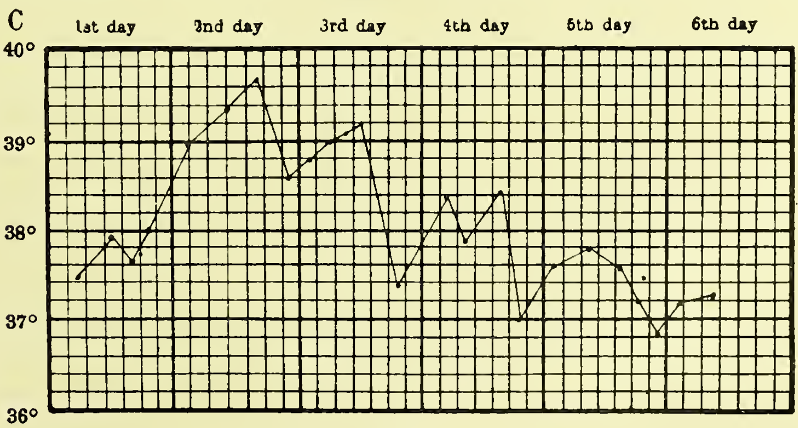


Type I.

assistant physician to the Municipal General Hospital at Friedrichshain, which appeared in the *Centralblatt für klinische Medicin* for January 11th, 1890.

Influenza shows a marked tendency to relapse, and to this feature the indirect fatality of the malady is largely due. In the epidemic of 1847 the death-rate was estimated at only 2 per cent of the cases observed in London. It was probably not higher in the several epidemics which have occurred since 1889 up to the present date (1900). In the winter of 1889 at least a fortnight elapsed between the occurrence of the earliest cases and the fatalities from influenzal bronchitis and pneumonia.

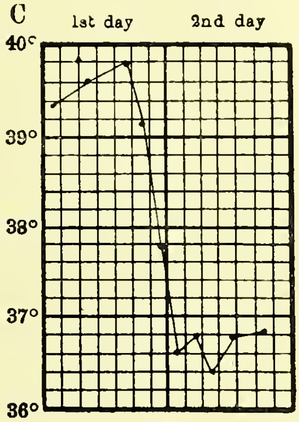
So far from establishing immunity, an attack of influenza seems to predispose an individual to a second or even a third visitation of the disease. In this respect influenza resembles erysipelas, diphtheria, and perhaps pneumonia, or "pneumonic fever."



Type II.

Atypical Forms

Apart from the very many cases which range themselves more or less accurately under the four types just described, there remain not a few cases of influenza which do not admit of being properly classified. They are atypical—that is to say, they conform to no type. With equal propriety such cases might perhaps be called polytypical, or many-typed.



Type III.

In the first place, as Zülzer pointed out in his classical monograph on "Influenza" in von Ziemssen's *Cyclopædia of the Practice of Medicine* (1875), a great number of rudimentary forms are observed in every epidemic. "A considerable part of the population in fact, under the influence of the 'genius epidemicus,' exhibits a state of indisposition which does not amount to a full febrile affection, but which is shown to be a general invasion of the system by slight coryza, by confusion of the head, by one's quickly becoming fatigued, by disinclination for business, and often by sore throat, tickling cough, etc." Zülzer further shows that it is seldom possible to bring all the special cases of influenza within the compass of the ordinary classification because the forms under which the disease manifests itself are made up of various combinations.

Lastly, the same author observes that the "features of the disease acquire still greater diversity from the various *complications*." Many

phenomena of the disease, especially the nervous symptoms, exhibit "important modifications according as they affect more or less irritable individuals; in hysterical and such other patients influenza often assumes a pronounced nervous or spasmodic character. In the case of hæmorrhoidal affections, and in the rheumatic or gouty diathesis especially, the muscular pains will occur. With young children symptoms of congestion of the brain are often seen, and this perhaps may be the explanation of the unfavourable result sometimes observed in their cases."

Perhaps also the form of the disease which Finkler has called "chronic influenza" should be included among the atypical or aberrant varieties. When Finkler asserts that influenza may assume a chronic character, he refers to the actual continuance of the influenza bacilli and to a resulting chronic bronchitis or chronic pneumonia. Various authors, among them Pfeiffer, Wassermann, Beck, and Kruse, have assured us that the bacilli of influenza may repeatedly be found in the sputum for weeks and months. In such patients cough and expectoration persist after the influenza has seemingly passed away; and, without any known cause, at intervals of a few days or a few weeks, the symptoms of a bronchial catarrh return, so that again and again cough and expectoration increase — to decrease in turn, probably without medical attendance or treatment. In these chronic cases of influenza, Finkler says he is prepared for everything which occurs in the acute form—sudden fever, neuralgia, attacks of perspiration, digestive disturbance, asthmatic symptoms, coryza, sore throat,—all these reappear without warning, alternating in such a manner that the patient often attributes the cause to a "change in the weather." Only a bacteriological examination —which should always be made—will distinguish such cases from a bronchial or pulmonary tuberculosis, or from a secondary diplococcus infection.

Effects on Different Systems

The statement made above that influenza seeks out the weak point (*locus minoris resistantiæ*) in an individual gives a clue to the multiplicity and severity of the complications and sequels of the disease.

The Respiratory Tract.—As was to be anticipated, the poison of influenza falls with special violence on the respiratory tract. In Dublin, during the first quarter of 1890, 973 deaths were referred to diseases of the respiratory system, exclusive of phthisis. This number was 34 per cent in excess of the average for the corresponding quarter of the previous ten years. The deaths from bronchitis were 654, against an average of 507, or an excess of 29 per cent. Those from pneumonia were 211, or 79 per cent above the average.

Foremost among the secondary affections which complicate influenza stands pneumonia, to which the heaviest death-roll is paid by the victims of the primary or influenzal infection. But, in this instance, the term pneumonia must be taken in a generic rather than in a specific sense. As a matter of fact, broncho-pneumonia, true lobar (croupous) pneumonia, and cellular (influenzal) pneumonia are well-recognised varieties of lung-inflammation which occur in or after influenza.

Leyden, in a communication to the Medical Society of Berlin, states that the pneumonias observed by him showed a peculiar course—severe pain in the side and dyspnœa were rarely noticed; the local process was not altogether typical; frequently it was necessary to watch for three or four days before any evidence of a localisation of the disease was forthcoming. Then a crepitating râle was heard over a wide area, and this perhaps the very next day would have disappeared to show itself in some other situation. Not very often a firm hepatisation occurred with clearly mapped out dulness. Again the typical sputum of pneumonia was often wanting. Bacteriological investigations revealed the presence of three kinds of microbes: (1) Diplococci, which represented the well-known pneumonia - diplococci of Fränkel; (2) Streptococci; (3) Staphylococci. Leyden adopted the view that the forms of pneumonia are different —typical genuine pneumonias with deviating course; mixed forms, especially in those combined with pleural effusion; lastly, simple streptococci pneumonias.

Ribbert, discussing the possibility of a causal significance of the *Streptococcus pyogenes* in relation to the phenomena of influenza, alludes especially to the inflammation of the lungs, whose peculiar erysipelas-like spread, on which Finkler lays so much stress, and whose anatomical relations admit of being referred back to the influence of the streptococcus. Ribbert points out that, in contrast to ordinary croupous pneumonia, the cut surface of the hepatised lower lobe in three cases presented an almost smooth appearance, the exudation was soft, very rich in cells, and poor in fibrin (hypinosis). Cultivation experiments with the tracheal mucus, the lung tissue, the spleen, and the kidneys, furnished in five out of eight cases the *Streptococcus pyogenes*, or else the *Streptococcus erysipelatosus* (which has been shown to be identical with the former), the presence of which microbe could be demonstrated in the sputum also of the influenza patients. His investigations on the whole yield the result that in all cases in which micro-organisms were at all capable of demonstration, the *Streptococcus pyogenes* was found. Only once was there in addition a coccus which had a great resemblance to the *Diplococcus pneumoniæ* and probably represented a modification of the same.

Finkler observed 45 cases of influenzal pneumonia, of which only 2 came under the description of typical lobar pneumonia, while the other 43 were regarded as cases of the disease which he has often described as "Streptococcus Pneumonia." Seven of his patients died, post-mortem examinations being made in three instances. He regarded the pathological condition as a preponderating cellular inflammation with participation of the interstitial tissue. The cellular nature of the inflammation, together with the pronounced tendency it exhibits to develop by spreading indefinitely, in Finkler's opinion justified him in describing this disease as an erysipelas of the lung. He points out that the resemblance of this form of pneumonia to erysipelas consists, not alone in the anatomical characters of the inflammatory process, but also in the fact that both diseases depend on the presence of streptococci. Finkler looks upon this streptococcus pneumonia as a localisation of the exciting cause of influenza in the lungs. As to this last point one would be more inclined to agree with Leyden and Levy that the question is much more one of a secondary infection, for which the influenza merely laid the foundation.

It is to be remembered that the foregoing observations were made in connection with the great pandemic of influenza in 1889-90, antecedent therefore to the discovery by Pfeiffer in 1892 of the *Bacillus influenzæ* in the purulent bronchial secretion, and by Canon in the blood of patients suffering from epidemic influenza. That discovery must certainly be regarded as conclusive proof of the existence of a specific primary infection of the lungs to which all other infections are accidental and secondary.

We may then conclude that Pfeiffer's bacillus itself produces a catarrhal bronchitis and a pneumonia, while the presence of influenza predisposes to secondary infections of the lungs by the *Diplococcus pneumoniæ* of Fränkel, the *Streptococcus erysipelatis* of Fehleisen, the *Bacillus pneumoniæ* of Friedländer, and possibly the *Staphylococcus pyogenes aureus* of Ogston.

The *pleura* is often involved in the primary or secondary pathogenic processes of influenza. Researches carried out by a Collective Investigation Committee in Germany showed that the pleura became engaged in from 12 to 33 per cent of the cases of pneumonia complicating influenza. At Riga Krannhals met with "exudative pleuritis" in 12 out of 220 cases of influenza. In the Riga hospital there were 7 cases of pleural effusion among 87 cases of influenza. The exudation was, as a rule, sero-fibrinous, but in 6 instances it was purulent. The affection may supervene upon bronchitis without pneumonia. The bacterial factor in its production is most frequently the *Streptococcus pyogenes*, but various staphylococci also play a prominent part in its etiology. Finkler of Bonn alludes to the intense dyspnœa which often attends the pleuritis of influenza — a dyspnœa which he rightly regards as not merely mechanical, but also probably toxæmic in its origin. Long ago Robert James Graves of Dublin gave a graphic description of the extraordinary dyspnœa witnessed in the influenza of 1836-37. According to him, "even in many cases where the bronchial mucous membrane is but slightly engaged, the amount of dyspnœa is remarkably great. Indeed, it might be said with much truth that the dyspnœa was by no means proportioned to the extent of pulmonary inflammation." Having pointed out that in many bad cases of influenza the dyspnœa is intermittent, Graves suggests that the respiratory derangement may depend on the same general cause which produces the whole train of symptoms, and that it might exist even where there was no bronchial inflammation at all. "The dyspnœa," he adds, "appears to be chiefly attributable to some impression made on the vital activity of the lung."

Influenza is a perilous complication of pulmonary consumption. In London at the height of the epidemic of 1889-90 the weekly number of deaths from phthisis rose to double the average. In Dublin phthisis caused 437 deaths in the first quarter of 1890, or an increase of 90 deaths, or 26 per cent, as compared with the average for the first quarter of the previous ten years.

So long ago as 1803 Dr Woodford of Almsford wrote: "When the influenza attacked subjects apparently predisposed to phthisis, it never failed to increase and call into action the latent seeds of that disease, and in several instances to induce speedily a confirmed state of it, which quickly proved fatal." In this opinion most students of influenza will agree, although there have been a few dissentients from time to time. We are thoroughly in accord with Dr. J. F. Goodhart when he says that in a large number of cases influenza lays its victim open to an attack of pulmonary tuberculosis. "Many a case," he writes, "seemed to start from an attack of influenza, and many a case of phthisis was certainly sent on its way with an alarming increase in the rapidity of its progress."

Finkler has drawn attention to the fact that in phthisical patients particularly influenza lasts an extraordinarily long time, that it becomes as it were chronic in them, and from time to time induces fresh inflammatory processes in the cicatrising tuberculous areas in the lungs.

Partly also through the damage inflicted by it on the elasticity of the lung, but chiefly from its hurtful effects upon the pulmonary nerves, influenza lays the foundation for bronchial asthma, a sequel which is usually transitory, but may be persistent.

Another distressing sequel, to which Professor Osler has drawn attention, is diffuse bronchiectasis.

The Cardio-Vascular System. — Influenza *poisons* the heart. These words perhaps will best express the disastrous effect which this strange malady has upon the heart muscle, but especially upon the innervation of the heart. Indeed, the term "heart failure," now on every one's lips, was scarcely ever heard before the pandemic of 1889-90. The very treacherous and dangerous form of influenza known as the cardiac type may occur with or without pneumonia. The frightful ebbing of strength in many cases of influenzal pneumonia in persons of middle life or advanced in years reaches its climax in a true heart-palsy. In other cases, uncomplicated with pneumonia, the heart collapses, sometimes with a suddenness that suggests rupture or cardiac thrombosis.

In Dublin the outbreak of 1889-90 had an immediate and untoward effect in raising the mortality from diseases of the organs of circulation, including "heart disease," in the most general sense of the term. A marked rise in the number of deaths coincided with the chief epidemic period, the second, third, and fourth weeks of 1890. In the fourth week diseases of the circulation were returned to the Registrar-General for Ireland as the cause of death in the Dublin registration area in 30 instances, compared with a ten years' average of 12·2 deaths for the corresponding week. Of the 30 victims, 14 were aged between 40 and 60 years, 9 between 60 and 80, and 4 were above 80 years.

In estimating the malign influence of the disease on this class of maladies, it is noteworthy that neither intense cold nor undue warmth in winter raises the death-roll from diseases of the circulatory system to any perceptible extent. This interesting fact throws into bolder relief the baneful results of influenza.

The grosser lesions of pericarditis and endocarditis must be accounted rare in or after influenza. Even evidences of myocarditis and acute degenerations of the cardiac ganglia and nerves are often sought for in vain, alike before and after death. Nevertheless, in a large number of cases the physical signs and symptoms bear testimony to a profound influence of the poison of the disease upon the myocardium and its nerves. Breathlessness on slightest exertion, coldness and blueness of the extremities, restlessness, the presence of blowing systolic murmurs at the apex, paroxysmal tachycardia, or arrhythmia of the pulse, epigastric pulsation —all point to acute dilatation of the ventricles —a pathological state brought about by changes (inflammatory or degenerative, or both) in the heart muscle and in the cardiac nerves. The pulse is not only rapid, irregular, or intermittent, but its rate varies remarkably with change of position, becoming extremely rapid when the patient sits up. This is what Huchard calls the *pouls instable.* The toxins of influenza may affect the blood-vessels, causing thrombosis or hæmorrhages, lesions which are strictly analogous to those which occur in the vessels in the course of other infective disorders. Nose-bleeding, hæmorrhage from the ear, hæmoptysis, hæmatemesis, bloody stools, hæmaturia, and menorrhagia have all been observed more or less often.

The Skin.—The skin may be the seat of various rashes in influenza. Thus in the hæmorrhagic variety of the disease petechiæ may occur. Or again, from vaso-motor paresis, fleeting erythematous rashes (erythema fugax) may develop, resembling either measles (morbilliform) or scarlatina (scarlatiniform). Papular sweat rashes, with miliary vesicles or sudamina, are also met with. Herpetic eruptions are especially common.

In contrast to dengue fever, however, influenza must be regarded as a non-eruptive fever. When the rashes just mentioned do appear, they are accidental rather than essential or specific, and they result from vaso-motor paresis, or hyperpyrexia, or profuse sweating, or from the ingestion of such drugs as quinine, phenazone and its congeners, or salicylate of sodium.

Erysipelas, eczema, acute furunculosis, and suppurative follicular impetigo have also been observed as complications or sequelæ. Pemphigus has occurred in rare instances.

Extreme dryness of the skin is a rare epiphenomenon. Extreme sweating (hyperidrosis) is not uncommon. Finkler has seen a man who suffered for months after influenza from hyperidrosis which nearly drove him frantic. When the man stood naked in a cool room the perspiration would literally pour in streams from his skin. It is interesting to note that in this case atropin had no effect whatever, while the attacks suddenly disappeared after salipyrin was administered.

The Kidneys.—Albuminuria is by no means uncommon in influenza, but nephritis must be looked upon as an infrequent complication or sequela. When it occurs it is a true acute infective glomerulo-nephritis—to use Klebs' term. Glycosuria has tolerably often been met with in or after influenza.

The Digestive System.—The digestive system suffers severely in many cases of influenza, but the disturbances are of functional or nervous origin rather than pathological in character. The thickly coated tongue, foul mouth, and fœtid breath have already been mentioned. There is sometimes absolute loss of appetite, amounting to disgust at the very idea of food, constipation, flatulence, vomiting, and less frequently diarrhœa. Occasionally tenderness on pressure and pain in the ileo-cæcal region indicate the presence of a true influenzal enteritis. If to these symptoms distension of the abdomen, insomnia, delirium, rapid pulse, and enlarge-

ment of the spleen be added, the diagnosis between true enteric fever of a severe type and the *enteric* or *typhoid* form of influenza becomes most difficult. Widal's test for enteric fever may help the physician to a correct opinion. Finkler points out that in these cases the anorexia and accompanying gastric symptoms may last for weeks after the influenza has declined, so that a notable loss of flesh and even a grave cachexia may take place.

The liver is rarely much affected in influenza. It may, however, be the seat of congestion or of a parenchymatous degeneration, such as occurs in other acute toxæmias. Marked jaundice is also rare, though an icteric tinge of the conjunctivæ is often seen. Dr. Bäumler of Freiburg met with slight jaundice so frequently that he regarded it as a valuable diagnostic symptom of influenza. Still he admits that he could not demonstrate the presence of bile pigments in the urine, and the "jaundice" was confined to a yellow coloration of the sclera.

The Nervous System.—"There is hardly a nervous symptom in existence," says Finkler, "which has not been observed in influenza." He proceeds to instance headache, backache, neuralgia, myalgia, hyperæsthesia and anæsthesia of the cutaneous nerves, vertigo, insomnia, delirium, syncope, debility, post-grippal neurasthenia, hysteria, and hystero-epilepsy, coma (so that the malady resembles the sleeping sickness or noma of the West Coast of Africa), chorea, singultus, yawning, tetanoid spasms, and local paralyses. For these various nervous phenomena Finkler believes that we must seek an explanation in the action of some soluble toxin. He further believes that they are all purely functional disturbances, basing the opinion on the facts (1) that the combination of nerve symptoms with atrophy was not sufficiently marked to justify a diagnosis of anatomical changes in the nerves themselves; (2) that recovery has frequently resulted in such cases with astonishing rapidity; and (3) that drugs such as phenazone, salipyrin, or quinine frequently caused a change for the better, or even a perfect cure so suddenly "that the diagnosis of a functional nervous disturbance was forced upon us *ex juvantibus*."

It must not be supposed that organic lesions of the nervous system are unknown in connection with influenza. Far from it. In the first place, this is one of the diseases of which the toxin damages the nerves. Peripheral neuritis, shown by pain, paræsthesiæ, and paralysis, may be present locally or as a multiple neuritis (peripheral polyneuritis). Or "nuclear paresse," as Leichtenstern well terms them, may arise from lesions of the motor nuclei of the third and fourth cerebral ventricles caused by the toxins of influenza.

Apoplectiform seizures have occasionally happened. In 1890 Leichtenstern reported eight instances, to which he afterwards added four more. The cerebral paralysis of influenza may show itself as a monoplegia or as a hemiplegia, and may be complicated by delirium, convulsions, and coma. This group of symptoms is probably due to microbic embolism of the vessels of the pia mater, similar to what may be observed in cases of epidemic cerebro-spinal meningitis and of ulcerative or malignant endocarditis. Fürbringer has reported two cases of grave organic cerebral affections following influenza, in which large hæmorrhagic foci in the brain were found after death. Similar focal diseases of the brain have been recorded by Bristowe, who observed five cases in which the preceding influenza was looked upon as the etiological factor in the encephalitic process (Finkler). In 1897 Pfuhl reported three cases of what he believed to be localisations of influenza bacilli in the brain. As these bacilli are now known to be capable of exciting suppuration, they may be the cause of primary purulent changes in the brain; or secondary purulent foci may result by metastasis from suppuration in the ear, frontal sinus, antrum, nose, or tonsil.

Purulent meningitis may be brought about in precisely the same way in or after influenza. At the same time Krannhals of Riga has observed a number of cases which clinically resembled an undeveloped cerebro-spinal meningitis, but which at the autopsy showed only excessive hyperæmia of the pia mater. In every such instance there was pneumonic infiltration of the lower lobes of the lungs. In quoting this observation, Finkler says: "It is of extreme importance to remember that we may have these marked meningitic symptoms, such as great fever, apathy, sopor, stiffness of the neck, strabismus, inequality of the pupils, grinding of the teeth, hyperæsthesia, bradycardia, and intermittent respiration, without any lesions whatever of meningitis."

Organic affections of the spinal cord are, according to Finkler, rare in influenza. Symptoms referable to a disseminated myelitis have been observed by Benno Herzog. Revilliod reports the case of a young girl who, after a relapse, became totally paraplegic, with symptoms of spastic spinal paralysis. The typical symptom-complex of Brown-Séquard's paralysis (hemiparaplegia with hemianæsthesia of the opposite side) was observed by Determann in a man aged 25. Acute ascending paralysis (Landry's palsy) has been described by Féréol and Laveran; acute anterior poliomyelitis by Teissier, Henoch, and Drashe.

Apart altogether from the brain disturbances, whether functional or organic, which have just been detailed, are the mental affections of influenza. The disturbances of the mind, or pyschoses, which attend or follow this strange malady, are countless in number and in variety. They form one of the most distressing, because one of the most intractable, features of the

disease. While it is true that serious mental disease, temporary or permanent, may supervene on any acute specific intoxication, such as typhus, small-pox, or enteric fever, yet influenza easily takes first place as a predisponent to psychosis. Nor is it only in those who are by heredity or by constitution predisposed to mental infirmity that influenza plays havoc with the mind. Dr. Julius Althaus examined the question of predisposition with great care, and found that there was a congenital or acquired neurotic tendency in 62·8 per cent of the cases of post-influenzal psychosis, but no such tendency in 37·2 per cent of such cases. Kirn divides influenzal psychoses into febrile and post-febrile, according as they declare themselves during the fever or after it has run its course. Delirium, attended by motor excitement, hallucinations, and sensory illusions, is the form which the febrile variety usually takes. States of mental exaltation and of depression follow each other in quick succession. There is insomnia. Recovery usually occurs, but the patient's memory may be a blank so far as his illness is concerned. The post-febrile psychoses, according to Kirn, are—acute mania, coming on immediately after the fever; melancholia and depression, appearing within a few days to a few weeks after influenza; and general paralysis, coming on as late as six months after the attack.

In addition to these graver diseases of the mind, we must note the not infrequent occurrence of either hysteria or hypochondriasis in the later stages of influenza. The following are extracts from a letter written by a victim of influenza in January 1900. The patient was a gentleman, aged about twenty-seven, who subsequently regained his usual excellent health. He writes:—

"As I am quite satisfied that the complaint I have (and many others) is something quite new in this country, I thought you would not mind my writing to let you know how matters stand with me. . . . I had a fair night's sleep—one or two 'heats and colds.' The next day I was not so well, and on Wednesday morning I went to Greenore Hotel. I walked on the shore after lunch, and had a very light dinner—fish and chicken. Having had some champagne at 4.30 P.M. I drank nothing at dinner, but took about a wineglassful of whisky punch at 10 o'clock. I slept the night through without any apparent changes of temperature, and felt as well as ever I did in the morning, when like *a fool* I took a tepid bath. This, I think, gave me a chill again, as, though I felt well all the morning and walked on the shore, I felt very dull towards evening. During the next night but one I was very dull and cold—nothing seemed able to warm me, and I got a bad attack of 'shivers.' By the way, when at Greenore, a young friend of mine was *wheeled* into the coffee-room of the hotel with all power of his legs gone, and he has had just the same symptoms as myself. . . . I need hardly say what a shock this gave me. . . . Some of the doctors looking after this case I mention told him to go out as much as possible; others told him to stay in—anyhow, the poor chap can keep no food in his inside, and cold is no name for him, though now and then he says he feels *hot*! This fever seems to me more like what fellows tell me they get at places on the West Coast of Africa—such as Bonny, Cape Coast Castle, and Lagos—for as far as I am concerned I have no sign of 'a cold' whatever. . . . If I am any way better to-morrow (Saturday) I will go home, as I am a bother here at night when I can't keep warm, even with a roaring fire. I hope you will not think this letter a great bother to read, but, as I said, I think these cases are new and interesting. Young M. of —— is another case just the same—a strong, hardy chap, and run down to nothing without any cold, and nothing to bother him in the world. . . . The only thing I am taking now are 'Carrara pills,' to keep me clear. Some of the cases seem to be taken the other way. My appetite is excellent, and especially for breakfast, for which I take porridge, boiled eggs (2), milk, and about a quarter of a cup of tea."

This letter illustrates very well the intensely subjective or self-conscious mental state of the convalescent from influenza. In a case recently under observation, a strong young fellow of twenty-two stated that he had never been well since an attack of influenza five months previously. His heart thumped at night, and flushes of heat would pass from time to time up his legs and thighs, finally affecting his head and face, and causing his temporal arteries to throb. These vaso-motor phenomena are very common, and cause infinite distress of mind as well as body.

Pathology and Anatomical Appearances

There is little that is distinctive or characteristic in the morbid anatomy of influenza. The disease kills through its complications and sequelæ, not by its primary effect on the tissues and organs. Hence the pathological appearances belong in a great measure to the secondary affections. Pneumonic consolidations in the lungs, which are often lobular rather than lobar; lesions of the pleura, of the bronchial tubes, of the myocardium,—all these are met with in many cases. But in the merely partial development of a specific pathology influenza closely resembles typhus fever. The chief feature is a widespread hyperæmia and catarrhal swelling of the mucous membranes lining the respiratory passages from the Schneiderian membrane to the bronchioles. Similar changes, though less marked and of less frequent occurrence, are observed in connection with the stomach and intestines.

The mucous membranes of the larynx, trachea, and bronchi show a redness of varying intensity (Ribbert), and, as a rule, are smeared with a tenacious grey or yellowish-green mucus. While these are the macroscopic appearances, Ribbert describes a very marked cellular infiltration of the mucosa, together with an engorgement of the blood-vessels,—these microscopic appearances giving one the impression of an erysipelatous process. This impression is strengthened by the fact that these catarrhal changes in the mucous membranes show a strong tendency to advance—spreading from the nasal passages and pharynx to the larynx and trachea, and bronchi, or in the reverse direction outwards from the bronchi to the larynx, pharynx, and nose.

In the lungs, the consolidation presents the appearance of splenification rather than of hepatisation. The cut surface of the pneumonic areas is usually smooth, wanting the minutely granular or granite-like appearance of a typical fibrinous pneumonia. Pfeiffer, as a result of his microscopical investigations, is of opinion that each infiltrated area of influenzal pneumonia stands in direct relation to a diseased bronchus. The epithelium of such a bronchial tube is infested with influenzal bacilli, which also lie in masses under the epithelium. The same observer has also found the purulent exudation in influenzal pleuritis filled with great numbers of bacilli.

As regards the heart, thrombosis within its cavities, endocarditis, and pericarditis are occasionally found. Weichselbaum particularly states that in a number of cases the myocardium showed a distinct fatty degeneration.[1]

The spleen, liver, and kidneys are not often profoundly altered in influenza.

As regards the nervous system, Jürgensen describes intense hyperæmia of the membranes of the brain. A. Pfuhl of Cassel states that he found the bacilli of influenza within the blood-vessels of the brain and spinal cord in every one of a series of cases showing congestive and inflammatory changes in the cerebral and spinal meninges. Foà of Turin found in a case of influenza, fatal by broncho-pneumonia and pulmonary hepatisation, numerous microscopic hæmorrhagic foci throughout the spinal cord, and in some places even degenerative changes. The hæmorrhagic areas were situated especially in the posterior horns almost invariably at the periphery, the degenerated areas for the most part in the lateral horns. The grey substance and the anterior horns were not in the least altered. Hetweg speaks of an influenza hyperæmia which affects the entire central nervous system. He regards the process as not an ordinary vaso-motor disturbance, but a forerunner of an inflammation.

[1] Finkler, *Twentieth Century Practice of Medicine*, vol. xv. p. 106.

Diagnosis

Influenza must be distinguished from dengue, acute lobar pneumonia or pneumonic fever, cerebro-spinal meningitis, measles, or other eruptive fever in childhood, and enteric fever.

Dengue fever is a tropical or subtropical disease. Its epidemics are circumscribed. The fever movement is much more decided than that of influenza. Complications seldom occur. An erythematous rash is of very constant occurrence. In contrast to dengue influenza is a non-eruptive fever. When rashes do appear they are accidental rather than essential or specific, and they result from hyperpyrexia, or profuse sweating, or from the ingestion of such drugs as quinine, or phenazone, or salicylate of sodium. In the epidemic of 1889-90, in addition to several cases of herpetic eruptions, the writer saw three cases of papular sweat rashes with sudamina, and one case with erythema fugax.

As to pneumonic fever, it is only in those cases of influenza which are complicated with pneumonia that any doubt arises. The diagnostics are—the strict lobar distribution of pneumonic fever, its entirely cyclic course, its viscid, rusty sputum, its critical defervescence. The features of influenza pneumonia have already been given in this article.

Professor A. Netter, of the Hôpital Trousseau, Paris, in his article on "Cerebro-spinal Meningitis" in the sixteenth volume of *Twentieth Century Practice*, points out that influenza in its nervous form presents certain symptoms which resemble very closely the symptoms of that disease. These are especially the pain in the head and the back, the general soreness, and not infrequently delirium and vomiting. The onset in both cases is usually equally sudden. Influenza may even be accompanied with suppurative lesions of the meninges as Eugen Fränkel observed in one case. To add to the difficulty epidemics of both cerebro-spinal meningitis and of influenza have coincided. Netter is persuaded that the two diseases have been confused a number of times, even in Paris, and that the cases of "grippal meningitis," upon the frequency of which several writers have remarked, were actually instances of epidemic cerebro-spinal meningitis. The diagnosis would turn on the presence of Kernig's sign of meningitis, and the result of a bacteriological examination of the cerebro-spinal fluid withdrawn by lumbar puncture, as advised by Quincke. Regard, lastly, should be had to the character of the prevailing epidemic.

Dawson Williams observes that influenza, if accompanied by much coryza, may resemble very closely the onset of measles, and the resemblance may be the closer if there be much depression and high temperature. The most characteristic single symptom of the prodromal stage of measles is photophobia. In a child

who has not previously suffered from measles the sudden onset of coryza, accompanied by dread of light, would raise a very strong presumption of measles, and it would be imprudent to hazard a suggestion of influenza even if that disease were prevalent. When the erythema of the palate (exanthem) appears early, it will be of assistance in confirming a diagnosis of measles.

In the epidemic of influenza of 1892, very many cases were observed by Forchheimer of Cincinnati, in which a differential diagnosis between scarlatina, rubella or rötheln, and influenza proved difficult at the beginning. After the cases began to increase in number the differential diagnosis seemed easier.

The diagnosis between enteric fever and the gastro-intestinal type of influenza will be aided by the application of Widal's test, and by Ehrlich's diazo reaction in a less degree. Attention should be paid to the prevailing epidemic. The presence or absence of rose-spots should be noted. The condition of the spleen should also be ascertained. The duration of the fever is greater in enteric fever.

Treatment

The *treatment* of the affection we have been considering is based upon common-sense principles. It is expectant, palliative, symptomatic. There is no specific for influenza; nor up to the present are there means at our disposal for securing immunity against its infection. The susceptibility to the disease varies remarkably from time to time in the same individual. It has already been shown in this article that immunity is not acquired by an attack, rather the reverse.

The *prophylaxis* of the disease turns on strict isolation, however difficult to secure. In the next place, the mouth and nose should be systematically and frequently disinfected by antiseptic inhalations, sprays, and washes. From his own experience in practice, the writer can speak most highly of quinine and resorcin among remedies of the class named. A grain of sulphate of quinine mixed with (but not dissolved in) a wineglassful of cold water makes an excellent and efficient aseptic gargle. The antimicrobic properties of quinine are well known, and its topical use after the manner just described at once relieves the symptoms of sore throat, which result from the strain of the phagocytic conflict in the tonsils—our first line of defence against the invading pathogenic microbes. Another excellent preventive is the frequent employment of a resorcin spray. Eighty grains of resorcin, dissolved in half an ounce of glycerine and seven and a half ounces of peppermint water or rose-water—such is the formula which has proved most useful as a mouth spray and also as a nasal spray. Inhalations of oil of eucalyptus, thymol, oil of mountain pine (*Pumilio*), and the like are also valuable aids in prophylaxis. Internally quinine has been given with apparent success as a preventive by Graeser, of the Polyklinik of Bonn. Finkler quotes Graeser's observations as follows:—

"In Bonn all the men of one squadron (of a regiment of hussars stationed at that place) were given 0·5 gm. (7½ grains) of quinine in 15 gm. (half an ounce) of whisky for a period of twenty-two days. During this experiment there became ill in the first squadron, 22 men; in the second, 7; in the third, 19; in the fourth, 42; and in the fifth, 32. The second squadron was that in which the quinine and the whisky had been given to the men. Out of the seven cases of sickness occurring in this squadron, three may fairly be excluded, because the disease made its appearance after the termination of the experimental period. The four remaining cases occurred on the second, fourth, fifth, and sixth day respectively of experimentation; after this period no more cases of influenza occurred in the second squadron while the quinine was being given, although the disease continued to progress in the others. This result cannot be due to chance."

During an epidemic of influenza in the winter of 1899-1900 in Manchester, the sanitary authority of that city, at the instance of their medical officer of health, Dr. James Niven, D.P.H., issued a placard containing the following "Precautions against Influenza":—

1. The sick should be separated from the healthy. This is especially important in the case of first attacks in a household.

2. Discharges from the nose and mouth should not be allowed to get dry on a pocket-handkerchief, or inside the house or workshop. They should be at once collected in paper or clean rag and burned. If this cannot be done, the paper or rag containing the discharges should be dropped into a vessel containing water.

3. Infected articles and rooms should be cleansed and disinfected. (*See* "Disinfection," vol. ii.)

4. Those attacked should not, on any account, join assemblages of people for at least a period of ten days from the commencement of an attack, as they are likely to convey the disease to others. In severe cases the person attacked should remain away from work for a period of three weeks from the onset of the disease.

5. During the epidemic special attention to cleanliness and ventilation should be shown in factories and workshops. Workpeople are advised to wear warm clothing and to avoid unnecessary exposure.

6. Persons who are attacked by influenza should at once seek rest, warmth, and medical treatment; and they should bear in mind that the risk of a relapse with dangerous complications constitutes a chief danger of the disease.

7. The attention of employers is especially called to these recommendations.

Dawson Williams, in an excellent article on "Incubation and Infectiousness," in *Twentieth Century Practice* (vol. xiii. p. 383), holds that the period of isolation ought to be a week to ten days after the commencement of the disease, according to the severity of the attack. In cases complicated by pneumonia it should be extended to the end of convalescence.

When a person is struck by influenza, only one course lies open to the patient, and that is, to take to bed with the least possible delay. Rest, warmth, and quiet are three sovereign remedies for the primary disease and the best preventives of its more deadly complications. We have said that there is no specific for influenza. True, yet many drugs play a useful part in its palliative and symptomatic treatment. Tonics, like quinine and strychnine; antineuralgics, such as phenazone (antipyrin), phenacetin, acetanilide; heart medicines, like digitalis, strophanthus, caffein; antiseptics, such as phenol (carbolic acid), salicylic acid, salol, salipyrin, all these and other similar remedies fulfil the several indications for treatment which influenza in its varying phases presents.

The most popular form for the exhibition of quinine in this disease is the ammoniated tincture, which may be given in one-drachm doses, equivalent to a grain of quinine, every four, six, or eight hours. After the first two or three days the official solution of quinine hydrochloride in tincture of orange peel may be given instead in like doses. Children take a grain of quinine mixed with and partially dissolved in milk without demur, especially if a little syrup of orange or syrup of orange flower water is added to each wineglassful of milk. Quinine salicylate may be given in wafer cachets containing three or five grains as required, where the combined effects of salicylic acid and quinine are desired.

In the heart-palsy and tedious weakness of convalescence of influenza strychnine is our sheet-anchor. In failing heart it is best given hypodermically—one twenty-second of a grain (= 5 minims of the official solution) night and morning, or every eight hours. In convalescence the following will be found an agreeable mode of administration:—℞ Liquoris strychninæ hydrochloridi ʒss., acidi hydrochloridi diluti ʒiss., tinct. aurantii ʒvj., aquæ chloroformi ℥vij. M. ft. mist. *Signa*: "One-eighth part twice or thrice a day with or after meals."

In this prescription dilute phosphoric acid may be substituted for the dilute hydrochloric acid, and tincture of quinine for the plain tincture of orange peel. Also, it is advisable to interrupt the taking of this mixture after a few days, should it cause headache or the spasmodic pains of strychnine-accumulation.

While hypodermic injections of strychnine, combined with morphia if the patient is restless or sleepless, tell best in the emergency of heart-palsy, digitalis, strophanthus, or caffein may be exhibited when the crisis of heart-failure is past. From two to five grains of caffein may be given every six hours in the more acute stage, a quarter of a grain of sulphate of spartein being added to each dose, if so desired. As caffein increases arterial tension, trinitrin, which so markedly reduces it, may (if necessary) be exhibited at the same time. But the use of the nitrites in cases of extreme weakness of the heart must not be indiscriminate or unwatched.

Camphor elixir, composed of spirit of camphor ten parts, syrup five, and distilled water one part, mixes well in water and proves an excellent diffusible stimulant, in doses varying from half a drachm to one drachm. An American writer, in the *Boston Medical and Surgical Journal*, recommends, in the crisis of the weak heart of pneumonia, fifteen minims each of tincture of valerian, spirit of lavender, and aromatic spirit of ammonia, every quarter of an hour, until the pulse shows that the heart is regaining tone and beating more efficiently. The external application of warmth over the præcordial region is often attended by the happiest results when the heart flags.

When choosing between digitalis and strophanthus it is well to remember that the latter acts more quickly than the former. It should not, however, be forgotten that a remarkable tolerance for digitalis is established in acute asthenic pneumonia; indeed, in some cases this drug seems to exercise an almost specific action on the disease.

Liquefied carbolic acid in 2-minim doses, three times a day, proved an advantage in the initial stages of influenza in 1890-91.

Salicylate of sodium, given in combination with effervescent citrate of caffein, is most useful as a mild aperient and in relieving the rheumatoid pains of influenza. Salicylic acid and phenol may be exhibited in the form of salol, or better still, salophen (Drewes of Hamburg). The dose of the latter compound is 10 to 30 grains daily, or even more. Another salicylic acid compound is salipyrin, which contains 57·7 per cent of that acid with 42·3 per cent of antipyrin (phenazone). This last-named coal-tar derivative is an invaluable antineuralgic when given in moderate doses. A favourite prescription is the following:—℞ Phenazoni gr. 12-20, tincturæ gelsemii ♏xx., aquæ chloroformi ad ℥ij. M. ft. haustus. *Signa*: "One-fourth part every second or third hour until pain is quieted."

A long experience leads the writer to the conclusion that phenazone even in very small doses is not a suitable drug for young children, in whom it produces cyanosis and other signs of collapse. Phenacetin is a drug of the same class which is safer, and which soothes to sleep while producing more lasting analgesic effects. It is but sparingly soluble in water, an objec-

tion which applies also to acetanilide (the so-called "antifebrin"), though to a less extent. A favourite and efficacious method of administering acetanilide is in the form of "antikamnia" tablets, which are found to contain about 70 per cent of that drug together with about 10 per cent of caffein and 20 per cent of sodium bicarbonate.

If the tormenting and sometimes excruciating pains of influenza are once relieved by one or other of the foregoing remedies, temperature is likely to fall, and the patient may be soothed to sleep. But a solemn warning is needed against any undue interference with the fever of influenza or any other febrile disease. The nature of the fever process is now far better understood than it was even a few years ago, and we have learned that "fever," or elevation of bodily temperature above the standard of health or "normal," serves a useful purpose, provided that it is properly controlled. There is, in fact, what the Germans aptly call "das Heil-Fieber"—"the fever which brings back health." At the close of an able address on "Antipyresis" before the Tenth International Medical Congress at Berlin, in 1890, Professor Arnaldo Cantani of Naples used the memorable words—"Das Fieber, das in so vielen Krankheiten der beste Verbündete des Arztes ist"—"the fever, which in so many diseases is the best ally of the physician." Fever, in a word, purges the system. Therefore, high temperature should not be unduly meddled with, particularly in influenza, when it so quickly spends itself, as a rule.

The dieting of the influenza patient will often test the physician's skill and tact far more severely than the mere prescribing of medicine. Of this disease it is especially true that there are no two patients whom precisely the same dietary will suit. We might say: *Quot homines, tot epulæ.*

Of course the dietetic rules which apply to any febrile disease apply equally to influenza. Slops at first, solids later on in a gradually ascending scale, from lightly boiled fresh eggs to chicken, sweetbreads, lamb's fry, trotters, tripe and cow-heel, fish, roast joints, etc. But there are cases in which even the lightest foods are spurned with loathing. Then water—cold or hot—may be sipped, or "egg water" (the *eau albumineuse* of the French) may be given. This excellent "dish" is prepared by blending with a pint of cold water the whipped-up whites of from two to four eggs, and flavouring with salt or cinnamon. White wine whey may be given with equal parts of unflavoured egg-water, if a stimulant for a very delicate stomach is desired. A more substantial meal is egg-flip. The lightest form of this is made by pumping soda-water from a siphon on to the whipped-up yolk of an egg. Then the animal broths may be given, always remembering that an almost infinite variety of flavour may be obtained by mixing different broths together—for example, chicken-broth and beef-tea in equal parts. In a word, in attempting to draw up a bill of fare for a fastidious influenza patient,[1] we should always remember that "what is one man's food is another man's poison." Dr. T. King Chambers, in his excellent *Manual of Diet in Health and Disease* (published in 1875), reminds us that when the tailor in Laputa sternly refused to take the usual measurements, and insisted on constructing Captain Gulliver's coat, waistcoat, and breeches on abstract principles, the customer vowed it was the worst suit of clothes he ever had in his life. Dr. Chambers adds: "We should certainly fail in the same way if we did not take the measure of numberless contingencies in the daily life, and numberless peculiarities in the persons of those who consult us about their diet and regimen."

Always true, these words apply with special propriety to the dietetic treatment of influenza.

Infra-.—In compound words *infra-* means *below*, e.g. *infraclavicular* (below the clavicle), *infracostal* (below a rib), *inframaxillary* (relating to the lower jaw), *infrascapular* (below the scapula), etc.

Infundibulum.—A funnel-shaped passage or depression. *See* PHYSIOLOGY, RESPIRATION (*Respiratory Mechanism, Infundibular Passages of Lung*); FALLOPIAN TUBES (*Anatomy, Ostium Abdominale*).

Infusa. *See* PRESCRIBING.

Infusion, Saline.—A solution of common salt in distilled water (1 drachm to the pint), used for injection into the subcutaneous tissue, a vein, or the rectum in cases of hæmorrhage, eclampsia, sepsis, etc. *See* LABOUR, POST-PARTUM HÆMORRHAGE (*Post-hæmorrhagic Collapse*); SHOCK.

Infusoria. *See* PARASITES (*Protozoa, Infusoria*).

Ingluvin.—A digestive preparation obtained from the crop of the chicken; fowls' pepsin; used in intractable vomiting and digestive disorders.

Ingravescent Apoplexy.—That form of apoplexy in which the loss of consciousness does not occur suddenly, but is gradually and steadily developed. *See* HEMIPLEGIA (*Condition at Onset*).

Ingrowing Nail. *See* NAILS, AFFECTIONS OF (*Unguis incarnatus*).

Inguinal.—Relating to the *inguen* or groin, *e.g.* inguinal artery, inguinal canal, inguinodynia (pain in the groin), inguinal hernia (*see* HERNIA), inguinal hydrocele, etc.

[1] See "Invalid Feeding."

Inhalation-Pneumonia.—Inflammation of the lungs (broncho-pneumonia) following upon the aspiration into the larynx and bronchi of particles of food or drink, as in cases in which the sensitiveness or the structural integrity of the parts concerned in deglutition is affected; aspiration- or deglutition-pneumonia; ether-pneumonia.

Inhalations.—Volatile drugs may be given as *vapores* or *inhalations*, and they may have an irritating effect on the bronchial mucous membrane (*e.g.* iodine, bromine, sulphur fumes, ammonia, etc.), or a stimulating action (*e.g.* creosote, cubebs oil, benzoin, etc.), or a soothing action (*e.g.* conium, hydrocyanic acid), or a disinfecting action (*e.g.* iodoform, carbolic acid, oil of juniper, etc.). *See also* PHARMACOLOGY, PRESCRIBING; BRONCHI, BRONCHIECTASIS (*Treatment*).

Inhalers. *See* ANÆSTHESIA.

Inheritance. *See* HEREDITY; HÆMOPHILIA; SYPHILIS; etc.

Inhibition.—The power of restraining action, whether exercised voluntarily or reflexly. *See* INSANITY, NATURE AND SYMPTOMS (*Personality and Subconsciousness*); INSANITY, NATURE AND SYMPTOMS (*Types of Episodic Insanity, Defects of Inhibition*); PHYSIOGNOMY AND EXPRESSION (*Expression of Brain Faculty, Inhibition*); PHYSIOLOGY, TISSUES (*Nerves, Inhibitory*); PHYSIOLOGY, CIRCULATION (*Intracardiac Nervous Mechanism*).

Inhumation.—Earth burial (*q.v.*).

Iniencephaly.—The teratological type

in which through the backward bending of the head upon the dorsal region the nape of the neck loses its external position and is enclosed within the somewhat globular mass formed by the head and trunk (Gr. ἰνίον, nape of the neck, and ἐγκέφαλος, brain); the occiput is imperfectly formed, and there is usually spina bifida of considerable extent. *See also* TERATOLOGY (*Malformations of the Spine*).

Iniodymus.—Twins having a single body with two heads joined laterally and posteriorly.

Inion.—The occiput or nape of the neck.

Iniopagus.—The variety of craniopagous twins (*i.e.* twins united by the heads) in which the union is by the occiputs.

Injections. *See* ANEURYSM (*Treatment, Injection of Coagulants or Ergotin*); CHOLERA, EPIDEMIC (*Treatment, Injection of Saline Fluids*); ENEMATA; INFUSA; SCROTUM AND TESTICLE, DISEASES OF (*Hydrocele, Treatment, Injection of Irritating Liquids*); SHOCK (*Treatment, Saline Solution*); SNAKE-BITES (*Treatment, Antivenine*).

Injuries. *See* MEDICINE, FORENSIC; UNCONSCIOUSNESS; *and under* the various organs and parts (BLADDER, ANKLE-JOINT, etc. etc.).

Innocent Tumours. *See* TUMOURS.

Innominate Artery. *See* ANEURYSM (*of the Neck*); ARTERIES, LIGATURE OF (*Innominate Artery*).

Innominate Bone. *See* LABOUR, PROLONGED (*Pelvic Deformities*).

Innsbruck. *See* THERAPEUTICS, HEALTH RESORTS (*Austria*).

Inoculation, Preventive. *See* IMMUNITY; PLAGUE; RABIES; SMALL-POX; THERAPEUTICS, SERUM THERAPY; VACCINATION; YELLOW FEVER.

Inoscopy.—Jousset's method of examining exudates for tubercle bacilli: first fibrin masses are allowed to form, and these fix the bacilli; then the fibrin is dissolved away (by means of a solution of glycerine, hydrochloric acid, and sodium fluoride), and the fluid is centrifuged; the bacilli are left in the sediment. It is not, however, an absolutely certain method (Körmöczi and Jassniger).

Inosite.—Muscle sugar (Gr. ἴς, ἰνός, a muscle). *See* PHYSIOLOGY, TISSUES (*Muscle, Chemistry of*); PHYSIOLOGY, FOOD AND DIGESTION (*Sources of Principles of Food, Flesh*); URINE, PATHOLOGICAL CHANGES IN (*Sugars, Inosuria*).

Inquest. *See* MEDICINE, FORENSIC (*Certification of Deaths, Procedure in England and Ireland*).

Inquisition.—An inquiry into the cir-

cumstances of a case, *e.g.* the mental condition of any person, when it is called an Inquisition of Lunacy.

Insanity.—The subject of Insanity is here discussed in the following four main sections:—

1. **Etiology.**
2. **Pathology.**
3. **The Nature and Symptoms.**
4. **Treatment.**

See further under Adolescent Insanity; Alcoholic Insanity; Anæsthesia, General Physiology (*Remote Effects*); Bed-Sores; Brain, Tumours of (*Symptoms*); Brain, Surgery of (*Concussion, Treatment*); Bronchi, Bronchitis (*Insanity of Cyanosis*); Chorea (*Choreic Insanity*); Civil Incapacity; Climacteric Insanity; Criminal Responsibility; Dipsomania; Epileptic Insanity; Fragilitas Ossium; General Paralysis; Hæmatoma Auris; Hypnotism; Lunacy; Lungs, Tuberculosis (*Complications, Mental*); Malingering (*Insanity*); Masturbation; Mental Deficiency; Morphinomania and Allied Drug Habits; Paralysis, Insanity associated with; Paranoia; Pregnancy, Affections and Complications (*Mental Complications*); Puerperium, Pathology (*Puerperal Insanities*); Senile Insanity; Sleep, Normal and Morbid; Stomach and Duodenum, Diseases of (*General Symptomatology, Remote Symptoms*); Suicide; Thyroid Gland, Medical (*Myxœdema*); Thyroid Gland, Medical (*Exophthalmic Goitre, Mental Condition*); Unconsciousness; Vice.

Insanity, Etiology of.

Heredity and Stress.—The causation of insanity is a highly complex question in detail, yet the broad fundamental facts may be narrowed to two comprehensive statements regarding heredity and stress. The inherent faulty nervous organisation of the individual and the stress of environment constitute the basis of our considerations in regard to this subject. How far these considerations may lead us even in a brief survey of practical import must be evident, as they relate to all the various forms of systematic knowledge which are subordinate to the master art of healing. In elucidating the etiology of insanity we have not only to specialise in the department of psychiatry, we have to pass beyond its limits, and regard the question from the point of view of general medicine, we have to investigate sociological conditions, and pass in review large questions of education, of training, of hygiene. In short, if the science and art of medicine is to be of value in elucidating the causation of insanity, it must reveal the past, predict the future, and so order that prevention shall minimise the cases presented for cure. Cure comes late in the day, we are confronted with an intolerable mass of confirmed mental disorders from which recovery is impossible. That well-worn adage *obsta principiis* is the watchword of the medical profession, and to the prevention of disease our energies must be directed. Is insanity, then, one of the preventable diseases? Can we stamp it out as typhus has been subdued by hygienic control? Not so long ago phthisis was permitted to ravage the country, yet to-day we have high hopes of altering its incidence altogether, we are even impatient that it has remained so long with us. True, there is no gross demonstrable bacillus at work in the ordinary case of insanity. We have to trace the history of failure beyond the individual, and to deal with a more complex causation; yet in a larger hope for future generations. Were it not so, our labours in research would be vain, our counsel untrustworthy and misleading.

Definitions, Legal and Medical.—Many definitions of insanity have been put forward, and these need not be discussed here. The all-important fact is, that the brain is the organ of mind, and that mental disorders are to be correlated with the nervous system, more especially with the frontal lobes of the cerebrum. We need not enter upon a discussion of the conceptions of mental action. At any rate it in some way coincides with intimate and subtle changes in the nervous system. Face to face with the practical administration of law certain definitions are requisite, and these have been modified from time to time as knowledge grows, until they have become propositions which fairly represent insanity in sociological relations. As to criminals who are insane, the law requires to be informed if they knew the nature and quality of the criminal act, and that it was wrong. That is being further developed by the question, "Could he help it?" If the crime under review were committed by a person who did not *know* what he was doing (and Sir James Fitzjames Stephen imports a metaphysical distinction into the word *know*), or if he could not help it, then he is regarded as legally insane. Or, if a person executes a will which can be shown to have been made under the influence of ideas repugnant to the common

sense of society, he is held to have been of unsound mind, memory, or understanding. Lastly, the law may inquire whether a person is capable of managing himself or his affairs, and evidence is led to show what his conduct has been. Should his conduct be, in certain measure, different from what is commonly held to be right and prudent, it will be held insane, and he will be placed under judicial guardianship. The law takes but little cognisance of trifling deviations from conventional usage, and its definitions are designed to meet cases of patent insanity. Medicine, on the other hand, is less easily satisfied, and a medical definition of insanity becomes prolix in the endeavour to prove comprehensive. Small deviations are often momentous in consequences, but the superficial observer is impatient of the "trivialities" of science. Yet, as the twitching of a finger may be the herald of gross cerebral mischief, so may a trifling irritability grow into a mental storm of intensity and duration. The little cloud like a man's hand foretells the deluge.

As the world becomes more critical of *conduct*, so is the alienist most observant of *conduct*. The insane person is antisocial, or rather unsocial. He does not play his part as formerly, he does not play his part in conformity with the rules of the game. Dr. Mercier has summed up the whole matter in "three factors—disorder of the highest nerve arrangements, disorder of conduct, disorder of consciousness; and in every case the disorder of consciousness includes disorder of thought and feeling, of self-consciousness, and of consciousness of the relation of self to surroundings."

A consideration of Cullen's definition of insanity shows how far we have advanced. He said: "Insanity is a lesion of the intellectual faculties without pyrexia and without coma." Although we still speak of the intellectual faculties our business as physicians is with the underlying physical constitution; and Cullen's definition fails as completely in regard to the stated lesion as in regard to the absence of pyrexia. We are not now concerned with mere imperfections of the intellect, our measure of insanity is the observed reaction of the individual in relation to his environment; if that reaction is abnormal, owing to nervous disorder, the person is insane, and it is this question of disorder consequent on disease which makes insanity an affair of medicine. The disease may be of the most varied character. It may be what we conveniently term functional, it may be a coarse degenerative process, it may be an arrest of development. And in consequence of these infinite variations, from the indefinite beginnings of abnormal action to the most marked depravation of mind, there are innumerable cases of difficulty, elusive and changeable. Thus we have to fall back upon the rule to consider each case on its merits—the circumstances affecting each have to be weighed. Further, for our purpose in elucidating the causes of insanity we have to take into consideration the neuroses which are indicative of nervous instability. We cannot in this connection exclude these "minor" disorders, such as neurasthenia or hysteria. The neurotic inheritance by transmutation may evolve as marked insanity. The seeds of degeneracy are evident, and are only too apt to germinate.

We have arrived, therefore, at the conclusion that nervous instability, nervous derangement, nervous degeneration, are the underlying physical facts of insanity; that the mental aberration is at least concomitant with these facts, and that we have to deal with the various forms of insanity on the same general principles which guide us in other departments of medicine, specialising, indeed, as may be requisite in the due observation of nervous phenomena, and giving all attention to the details of conduct which constitute the reaction of the individual whose mental condition is the subject of investigation.

Pathological Considerations.—The anatomical, physiological, and pathological considerations which call for special study need not be enlarged upon here (*see* p. 451). Suffice it to say that these are of the utmost importance, and that the present state of knowledge is advancing rapidly year by year. We may, however, but briefly indicate that the nervous system is liable to disease in conditions similar to those occurring in other parts of the body. If there be a difference it is in the far-reaching effects of toxic agencies, traumatic accidents, inflammatory and degenerative processes affecting the delicate and complex constitution of the most highly organised and latest developed material in nature. The neurons with their complicated connections are susceptible of damage in measure yet undetermined by our finest instruments of research. Grosser changes, indeed, have been detected where, but lately, changes were denied. The territory of functional and idiopathic disorders is being rapidly investigated, and pioneers are charting the unexplored. We are thus enabled to comprehend the fatuity of advanced epileptic dementia, and the hopelessness of recovery, long recognised, is explained by the irreparable damage to the nervous elements. We are thus impressed by the primary importance of physical conditions which eventually become manifest in mental disorders. The mental disorder, the insanity, is not the main question in the mind of the physician. The grandiose speech and the extravagant conduct of the general paralytic are merely symptoms of a special cerebral disease. They do not constitute the real malady, however helpful in diagnosis. The general paralytic may be depressed and melancholic, but he is still

suffering from general paralysis. The essential nature of his trouble is the cortical disease of his brain. And so it is with the whole group of nervous diseases, we may be struck by the grotesque gestures of chorea, we may be appalled by the convulsions of epilepsy, we may be observant of the attitude of stupor, but we must pass beyond these apparent phenomena to attain a true understanding of the fundamental and real disorder. We must not be over-much occupied by the consideration of the mental symptoms of insanity, we must search for their causes in the ancestors of our patients and in the life-history of the patients themselves.

Causes stated.—The Scottish Commissioners in Lunacy have hitherto refrained from reporting upon the causation of insanity on the ground that the information obtainable is altogether untrustworthy. The English Commissioners, however, compile tables in regard to causation from information verified by the medical officers of the asylums. These tables show the percentage to the yearly average number of patients in the private and pauper classes admitted. During the five years 1892-1896 the table stands thus:—

	Private.		Pauper.	
	M.	F.	M.	F.
Domestic trouble	4·5	10·7	4·2	8·8
Adverse circumstances	8·3	3·9	7·2	4·5
Anxiety and worry	14·0	11·0	4·9	5·0
Religious excitement	1·7	3·1	1·4	1·5
Love affairs	1·2	3·6	·6	2·0
Fright and shock	·7	2·0	·8	1·7
Intemperance in drink	20·5	8·6	21·7	8·9
Sexual	2·3	·4	·9	·6
Venereal disease	3·9	·3	1·6	·5
Self-abuse	4·0	·9	1·8	·1
Over-exertion	·6	·6	·3	·2
Sunstroke	3·5	·2	1·5	·1
Accident or injury	2·9	·7	4·9	·9
Pregnancy	...	·7	...	1·2
Puerperal	...	6·5	...	6·4
Lactation	...	·8	...	1·6
Uterine and ovarian	...	2·4	...	·8
Puberty	·5	·9	1·0	1·0
Change of Life	...	7·4	...	4·5
Fevers	2·4	1·8	1·3	·8
Privation	·2	·5	1·5	1·5
Old age	3·6	3·8	6·5	7·3
Bodily diseases	9·0	10·9	14·5	13·0
Previous attacks	15·4	23·5	16·7	22·0
Hereditary	20·9	29·0	20·7	25·7
Congenital	5·2	2·0	5·1	3·9
Other causes	1·7	1·2	1·0	·7
Unknown	13·3	9·3	18·4	16·1

These "causes" include varieties of stress consequent upon which the patients under cognisance of the Commissioners succumbed to mental diseases, as well as the enumeration of cases of an hereditary nature. It will be seen that the mental strain imposed upon the individuals told more heavily upon those of the private class than upon those of the pauper class. The domestic troubles, as might be expected, fall heavily on women, while men are more oppressed by business anxieties and worries. But these single "causes" by no means represent the whole truth. Trouble and worry are involved with many other disadvantageous circumstances, and issue in a deteriorated state of health which leads by easy transition to mental disorder. Turning to the physical causes, the preponderating influence of alcoholic intemperance is at once recognised, but one may doubt if the figures represent a close approximation to the truth. A more rigorous investigation materially reduces the percentage, but when the social effects of alcoholism are considered, we may place against that reduction the cases of domestic trouble and adverse circumstances consequent on the abuse of alcohol. On an examination of 48 alcoholic cases out of a total admission number of 520, the proportion was found to be 9·2 per cent at the Perth Royal Asylum, which deals only with private patients. These divided into classes as follows:—A. Alcoholic persons without other ascertained cause. B. Alcoholics who were hereditarily insane. C. Alcoholics with alcoholic heredity. D. Alcoholics consequent on physical stress. E. Alcoholics consequent on mental stress.

A	3·26 per cent.
B	3·07 ,,
C	2·11 ,,
D	0·58 ,,
E	0·19 ,,
	9·21 ,,

Or to put a concrete case. A gentleman, whose mother had laboured under recurrent mania all her adult life, informed me that he had for years averted periodical attacks of mental depression by a free use of alcohol. On his admission his symptoms pointed to alcoholic intemperance, but the ordinary motor symptoms soon passed off, leaving him in a state of acute melancholia. Alcohol had at last failed him. His alcoholism was the result of insanity, and by no means the cause of it. There are other facts, however, which demonstrate the wide-spread influence of alcoholic intemperance in the production of insanity, *e.g.* the well-known observation of Dr. Yellowlees who showed how good times and high wages filled the Glamorgan County Asylum as a direct consequence of excessive drinking, while the converse also held good.

By this table of the Commissioners the effects of bodily diseases, toxic states, crises of life, old age, are also shown approximately. It will be seen that many had previous attacks of insanity in the proportion of 15 or 16 in men to 22 or 23 per cent in women. Lastly, we may remark the percentages stated in regard to heredity, which are assuredly understated owing to the difficulty of obtaining information as to the intimate family history of the cases coming under observation. At any rate, of patients received into the Perth Royal Asylum during the past eight years, the average proportion of those who had

an hereditary tendency to insanity is 43 per cent, and to neuroses 19 per cent in addition. And yet this is not all the truth, for in spite of every care and repeated investigations there are patients of such a nature that their hereditary taint can be recognised in the symptoms of their insanity, but who are excluded from the list of predisposed either by the wilful untruthfulness of their relatives or by the absence of definite information.

Heredity and Neuroses.—We shall now consider in some detail how this chief cause of insanity occurs. The outstanding laws of heredity are well known, and have been the subject of much discussion of late years. Darwin and Weissmann have each their disciples, and with regard to the main fact they are agreed that there is a transmission of parental attributes. It is sufficient for our purpose to accept the laws of heredity in the Darwinian form, for we cannot but believe that slow and gradual modifications by environment are transmissible (*see* "Heredity," vol. iv. p. 172). It is nothing to the purpose to search for the transmission of imperfect limbs consequent on parental amputations. The law of solidarity (the transmission of ancestral characters intact) repels any such objection, which is merely a revival of early objections to the Darwinian theory of evolution. But, although the tendency is for the child to inherit every character of the parents, there are various modifying influences at work, such as prepotency. There are also toxic influences, such as syphilis and alcoholism, which are undoubtedly transmissible and alter the course of the life of the offspring. There can be no doubt that the child of parents advanced in life is endued with less vitality than the normal, and that pathological heredity follows the same line as physiological heredity. We recognise that it is not so much a transmission of pathological characters as a predisposition to succumb to pathological influences. The child is not born phthisical, but its constitution develops into a condition favourable for the growth of the special bacillus. Then, again, there are well-known laws relating to consanguinity and exsanguinity, which must be observed if heredity is to be maintained in the most favourable condition as regards offspring. This becomes a question of practical importance in the consulting-room, for it is occasionally asked whether a person with an hereditary predisposition to insanity should marry. The marriage of first cousins is not uncommonly proposed in families of insane heredity, for these individuals have an undoubted tendency to form such connections. These marriages should be forbidden if a common and near ancestor has been insane, or if there is a near inheritance in each of the separate families; but it is permissible if insanity has declared itself in only one of the separate families. The question of inbreeding has not been discussed with moderation at all times; no doubt if continued for generations it is disastrous; but Huth has shown that arguments and facts are wanting to prove that consanguineous marriages in healthy families cause insanity and similar evils.

Insanity is a retrogression, a dissolution. It is a reversal of evolution, and as such has its beginnings in the latest evolved structures and functions of the brain. The recently acquired characters are least stable; they have been added in course of generations by the operation of natural selection and the conditions of environment. It has been said that if men gave the same attention to the propagation of their species as they do to the breeding of horses the race would have been more highly civilised, more highly developed physically and mentally. But while that is in measure true, we must recognise that the successes of the horse-breeder are few compared with his disappointments. We are in haste to be perfect, but the operations of the laws of heredity are not abrogated on that account. And the fact remains that marriages are not entered upon with that sense of responsibility to offspring which is desirable.

Degeneracy. — What is degeneracy? Much has been written round about this subject, and we have had elaborate descriptions of the stigmata of degeneration which are held in light esteem by those who are definitely opposed to the extreme teaching of Lombroso and the pamphleteering of Nordau. Following Morel, and making a special study of the prisoners under his care in Perth, Bruce Thomson came to the conclusion that heredity was the prime factor of criminality, and that environment determined the almost inevitable issue. He outlined the physical appearances of criminals, called attention to their malformations of body and perversions of mind. These are the stigmata of degeneration familiar to those who are brought into contact with the criminal and the insane. They are pathological deviations from the normal type, not to be declared morbid separately. The weight of evidence declares them to be morbid, just as in the Tichborne case Orton was not found to be an impostor on one count or another, but consequent on the summation of all the counts, by the strength of the condemnation of all the evidence. It would indeed be surprising if mind did not betray weakness when manifested in a body stunted, malformed, and weakly in organisation. The nervous system is imperfect in development, and mentalisation is degraded in proportion. One would not expect a perfect mind from an organism thus morbid in constitution. The reactions of the criminal mind are morbid in proportion as the criminal is degenerate. We do not desire to push this conclusion too far, holding that it is but a proportion of criminals who are degenerate; but when that proportion can be shown to exist in prisons and in asylums we

must recognise the facts, and endeavour to assess the amount of degeneracy when called upon in courts of law. For instance, in the case of Lawrie who committed a murder in Arran, insanity could not be proved; but he was declared to be so far degenerate or abnormal that he should not be hanged; yet he was sufficiently master of himself to undergo the modified punishment which was awarded him.

This morbid heredity, however it has arisen, is transmissible from parents to offspring. It may not be a certain form of insanity—it is rather a certain morbid conditioning of the nervous system which may declare itself as a neurosis in one generation and as a typical insanity in the next. The neurotic alcoholic is likely to propagate epilepsy, and the children of a hypochondriac may suffer from suicidal melancholia. Indeed, there is often a similarity in the very form of the insanity transmitted, even in the mode of suicide in successive generations.

It will be seen, then, that heredity may be altered by careful or by fortunate selection, the morbid constitution may be improved by the prepotency of healthy new stock.

The causes of degeneration manifested in neuroses and insanity are not far to seek. Briefly, we may group them as faults of inheritance, of civilisation, of alimentation, and of toxic origin.

We have seen how the laws of heredity are illustrated in the production of insanity. In conformity with the general observation, we remark that exhausting diseases in parents are followed by a weak offspring, and in degenerates there is a failure to develop shown by harelip or other monstrosities.

The instability is the heredity, and the faulty nervous system is the first cause. The more we study the intimate family history of each insane patient the more convinced we are of these antecedent circumstances. In the incidence of influenza now so commonly assigned as a cause of insanity, there is hardly a case to be found where hereditary defect cannot be traced. Where, then, did this evil begin? Is it a mere variation transmitted by direct inheritance? We believe not. In the mobile and varying circumstances of human life, in the progress of the race through the centuries, there must be a certain proportion of human beings who fall below the normal standard. By the mating of these, their generations successively become more unfit for the struggle for existence. In sub-human groups they would disappear at an early stage, but we are so accustomed to the methods of civilisation that we are apt to forget this. In the degenerative tendencies of mental disease we see families extinguished, the last survivor dying in the asylum. It is the conservation of civilisation which, so far, preserves those who are unfit, and we shall have yet to consider whether civilisation has its remedy.

Civilisation no doubt has a share in the production of insanity, but it is far from certain if that share can be estimated. The negroes of the United States of America were remarkably free from insanity under more primitive conditions. That immunity has disappeared, and they are now to be found crowding the asylums of the Southern States. But there are definite agencies at work in this unfortunate state of matters. Tubercle and syphilis have found a nidus in the negro, and he is suffering degeneration on the physical side in proportion to the mental degradation. Here we have, within a few years, an object lesson as to the effects of heredity on Darwinian principles. The germ cells have been affected, and in spite of Weissmann's declaration that they are apart from the soma, the results of a vicious civilisation are apparent. For civilisation has its vices and defects, and our boasted attainments and superiority over the "dark ages" are in some respects expressions in an optimistic vein. In its train civilisation has brought crowded cities, the factory system, squalid homes, and an ill-regulated population; or, at the other extreme, an unhealthy plutocracy, with a vacant, idle, and irresponsible life, craving for daily excitements, and gambling health away in the hot pursuit of pleasure. The benefits of civilisation cannot bribe us to shut our eyes to these unseemly facts, but should rather induce us to avoid the perils of our time, and to improve the conditions of the race. It is unlikely that ancient civilisations held a better place in respect of human life and happiness, or that the causes now operative in the production of insanity were formerly unknown. What concerns us more is the question as to whether insanity is really increasing in western civilisation, and specially if the increasing burden is as alarming and unpromising as it appears at first sight to be. In some forty years the number of insane persons coming under the cognisance of the Lunacy Departments of England, Scotland, and Ireland approximately expanded by 70,000, 9400, and 6200 respectively. These are figures which seize the popular imagination, and raise questions as to the possibility of stemming the rapidly rising tide. We cannot but believe that there is some increase of the occurring insanity as well as a vast increase in the registered insanity. Dr. Ireland has shown that the number of deaths from diseases of the nervous system in Scotland has grown by about one-third since 1855. No doubt much of this increase may be explained away by a better system of registration and wider knowledge, just as it may be argued that the increase in insanity and its allied conditions is indicative of an evolutionary advance with which weaklings cannot keep pace. The question has lately engaged the attention of the State departments in lunacy, and their general conclusion is that there is no alarming increase.

They have not arrived at this conclusion without painstaking inquiry, which showed that there were many points to be considered besides the simple fact of so many more insane persons to be reported upon.

Of course the vast proportion of these cases is classed as *paupers*, but it must be remembered that this is a most misleading statement. Mr. T. W. L. Spence has shown that only one-fifth of those so classed in Scotland really belong to the pauper class. It is in the nature of insanity to degrade not only in mentalisation, but also in social condition. Still it is true that where there are most paupers there are most insane, and it has been shown that if the larger number of cases come from the manufacturing towns, that number is balanced by the accumulation in the agricultural districts, where there is more stagnation, where the insanity is less curable, whence have been drained the more active spirits. The destruction of the poor is their poverty; the recklessness, the thriftlessness, the drunkenness of the lower classes involves instability of nerve tissue. The evil is apparent, and the remedy must come by an adequate appreciation of the laws of health and a different mode of life for the people.

Yet insanity is not entirely an affair of poverty and low life. The upper and middle classes have their burden, but in the general improvement in moral tone and the fuller appreciation of the benefits of mental and physical hygiene the causes are somewhat different. The direct cause of first importance in this respect is the over-strain to which men are exposed in the whirlpool of modern life. We may accept Dr. Farr's calculation, that the proportion between the upper and the middle classes on the one hand and the lower classes on the other is as 15 to 85. Certain it is that those occupations which are inseparable from mental stress have acquired a common notoriety in the production of disorders of the nervous system, and when to these is added a life of excess and dissipation, the result is a vast increase in cases of general paralysis, which is a disease of great cities and of great excess.

Turning to the next main cause, which is almost inseparable from other considerations, we may make but brief mention of malnutrition, starvation, and privations generally. It is a truism that the brain requires healthy blood in abundance. Whatever interferes with its due nutrition must issue in disorders of the nervous tissue more or less severe.

Toxic action of various kinds claims a more detailed notice. We may venture to divide these toxic agencies as follows :—

1. Exotoxic—alcoholic, plumbic, anæsthetic, cocaine, morphia, pellagra, ergot.

2. Autotoxic — rheumatic, choreic, gouty, diabetic, myxœdematous and cretinoid, uræmic, etc.

3. Microbic (?) — syphilitic, phthisical, influenzal, malarial, septicæmic.

It is manifest that these various degenerative causes would require a much more extended notice than can be given here, and we must refer to other articles in order to clear up the many points of interest. Broadly, in these various agencies we have something which is capable of inducing the degenerative conditions of the nervous system, to which we have already directed attention. Take exotoxic forms, such as alcohol and lead, the results of chronic alcoholic intoxication in the destruction of the chlorophil elements of the nerve-cells is matter of common knowledge. Lead has been demonstrated in the brain tissues consequent upon plumbic poisoning. The poison is virulent enough to destroy, and the enfeebled parents produce an enfeebled offspring.

Again, the autotoxic results of rheumatism, gout, etc., setting free malignant substances in the system by excess, or by derangement of metabolism, are familiar to all. Or, when necessary substances are deficient, as in myxœdema, the bodily welfare is threatened by deprivation. Insanity is thus caused by a defect as well as by an excess, and some of the most remarkable cures of cases of myxœdema have been recorded in asylums, where to the ordinary physical degeneration and infiltration a marked hebetude of mind, with delusions more or less pronounced, have been observed. The administration of thyroid substance rescued these sufferers by replacing in their systemic circulation that which had been wanting.

We have ventured to class syphilis, phthisis, influenza, and septicæmia as microbic agencies in the production of degeneration. In Russia syphilis is generally held to be a practically constant factor in the production of general paralysis, and there is in this country a growing tendency towards a similar conclusion. In every relation of physical condition syphilis is a powerful agent in degeneration of tissues, and we cannot but assign to it a very prominent position in this list. The other agents named are appropriately dealt with under their respective headings.

We thus arrive at the conclusion that there are very various and powerful physical influences at work in the production of degeneration, consequent on which we have manifestations of the various neuroses, the insane diathesis, the various forms of mental derangement, and the stunted development of idiocy in its various degrees.

Direct Causes.—Turning now to the more direct causes of insanity, which have been already referred to, we find that they are divisible into two main classes—those commonly spoken of as moral (mental) and physical.

The mental causes are the various kinds of mental stress to which men and women are ordinarily subjected. In practice it has been

found that cases of insanity are referable to these classes—adverse circumstances, anxiety, worry, domestic trouble, fright and nervous shock, love affairs (including seduction), over-pressure and over-strain, and religious excitement. After what has been said little remains to be added in elucidation of these headings. It is not so much steady, hard work with intervals of relaxation that is the cause, as the worry and scurry of life, the anxiety as to means of livelihood, the real troubles of existence. The excitement of politics leaves little trace on asylum records, while a financial crisis surely sends its victims to asylum care; and although religious excitement does not figure largely in the statistics of insanity, yet many cases occur showing delusions tinged by the religious movements of the time.

Passing to physical causes—what has been said elsewhere of alcoholic excess need not be repeated. Excess of sexual nature, over-exertion, malnutrition, and toxic agencies are all regarded as fertile exciting causes. The physical stress is apparent in such cases. We also find cardiac lesions in a great proportion of cases admitted to asylum, directly correlating rheumatic degenerative lesions with nervous deterioration. Patients with cardiac incompetence also occur through over-exertion in athletics, affecting the nutrition of the brain directly. The lesions of epilepsy, general paralysis, organic diseases of the brain, are referred to elsewhere, and their connection with this subject is evident.

We have still to consider the results of peripheral nervous irritation in the production of insanity. Schroeder van der Kolk directed special attention to what he called sympathetic insanity. He found that a great number of his patients suffered from constipation and were relieved by suitable treatment. He therefore laid the blame of their mental troubles on the peccant great intestine. That view was of importance in directing attention to the physical condition generally, but it does not seem that it can now command any considerable amount of support. The constipation is rather attributable to the defective action of the nervous system, and is a mere symptom of a more widespread and basic disorder. Similarly, recent attempts to correlate the insanity of women with uterine and ovarian disease can meet with no marked approval. The modern and probably correct belief is that the insane manifestations are due to disorders of the brain, and while it is very likely that certain of these disorders are due to toxic influences—perhaps owing to intestinal fermentation, or to deficient action of the ovaries—it would be absurd to confine our attention to the treatment of symptoms of peripheral irritations. We would not be misunderstood; the physical condition of every insane person demands the most searching investigation, adequate treatment of every departure from healthy action is imperatively required. While that is so, however, our later knowledge of nervous processes forbids us to lay stress on the peripheral conditions to the exclusion of the central mischief. And, further, these visceral neuroses must be studied in less complicated conditions if we would be successful in dealing with them in the insane. Professor Clifford Allbutt's lectures on this subject should be the book of reference in clearing the mind of specialism, and entering the freer air of general medicine.

There are also such causes as traumatism and sunstroke. These are of sufficient importance as to demand separate consideration. Dr. van Gieson has demonstrated by microscopical methods a very similar morbid process in the brain cells in both of these conditions. The shock is sudden and nutrition is immediately and gravely affected. We cannot, however, but entertain doubts as to statistics of the incidence of sunstroke, for a case who has been resident in tropical climates is never sent to an asylum without sunstroke being alleged as the cause. We would venture to suggest that malaria is far more efficient and common in this relation than sunstroke.

Again, in the evolution of the individual critical periods of life occur, and the stress of these periods tells heavily upon those who are burdened with a nervous inheritance. Puberty, adolescence, the climacteric, have each their special dangers, and it is in accordance with the laws of heredity that those most seriously affected by degenerative conditions will be earliest affected by insanity. Senility, on the other hand, if not premature, is the natural expression of failure of the brain after the exhaustion of a long life.

Yet again we observe a critical and exhaustive, if not a toxic condition supervening upon pregnancy, parturition, and lactation. These varieties of stress are familiar to every medical practitioner, and there is undoubtedly a disposition to minimise or overlook neurotic manifestations in connection with reproduction, a disposition which is a danger to doctor and patient. The physician should be urgent to guide matters aright when these abnormal signs first become apparent.

There yet remain two rather rare but interesting causes of insanity, viz. communicated and epidemic. Much has been written in reference to these cases, which have acquired a new and increased interest in connection with the later developments of our knowledge of hypnotic states.

The other considerations relative to the incidence of insanity might rather be held to fall under a statistical inquiry. The heaviest rate of occurring insanity falls on the years 35 to 45 in this country, but in the United States of America the greatest liability appears to be from

20 to 30. The question of sex is complicated by various considerations—the tendency being towards a slight increase of female cases, yet leaving the proportions nearly evenly balanced. With regard to condition as to marriage it may be concluded that, at marriageable ages, and in proportion to the population, considerably more married than single and widowed persons are admitted to asylums.

Prodromes.—This article would fail in practical value if some account of prodromes and prophylaxis were omitted. We shall therefore proceed to discuss the results of causation in their general and earlier manifestations, and give some indications of the measures which may be adopted with hope of success.

The penalties paid for inattention to the laws of health are heavy and various. They are not only exacted from the transgressor, but also from his progeny. The prodromata are of special interest, and demand early recognition and prompt treatment. Dr. Clouston has devoted his attention to the neuroses of development, and has set forth his observations in the Morrison Lectures for 1890, and separate articles will be found in this *Encyclopædia* dealing with epochal insanities. These may be briefly referred to as adolescent, climacteric, and senile. But the insanities of childhood and the neuroses of early life also demand attention, and preventive medicine must be called into action.

These symptoms are forebodings of nervous instability likely to eventuate in serious mischief, *e.g.* sleeplessness, headache, inability to perform mental work, loss of attention and self-control, unusual irritability, crazy reckless conduct, morbid fears and indecision. The nervous child is often precocious, a victim to night terrors, subject to convulsions consequent on derangements of digestion or the peripheral irritation of intestinal worms, or the troubles of dentition. The caprices, explosive rages (fulminating psychoses), and similar incidents, are often made light of by parents who, being themselves neurotic and unstable, are singularly unfitted to manage their own children. The morbid arrangement of the nervous fabric is forced upon our attention, the motor manifestations are evident, and conduct becomes so abnormal that it is hardly to be overlooked by the most superficial observer. These are assuredly indications for treatment, mental and physical. Separation from injudicious influences, the educational provision of a competent nurse of equable mind and adequate training, association with other children of normal development, are imperative. Here is a wide sphere for the employment of women, in educating and training children of self-centred, ill-regulated habits before these habits become fixed and unalterable. And the association of these children with others, so as to develop normal feelings with normal social instincts, is hardly less important. We must ever remember that these children are not merely nervous, but that in their hereditary misfortunes such facts as tuberculosis, gout, cancer, and other maladies may have a place. It is only of late that the cardiac diseases of childhood have been sufficiently recognised even amongst the leaders of the medical profession, and every asylum physician is familiar with the fact that cardiac complications are to be found in a very large proportion of the cases he receives for treatment. This wise guidance is a duty laid upon general practitioners; the specialist is called in too late when a definite malady is declared. We must look to the family doctor to direct the parents in their duty towards their children. That opens larger responsibilities than the apothecary dreamt of, but it is in strict accordance with modern tendencies, and it is thus that "doctors will become," in Gladstone's memorable saying, "the future leaders of nations." Year by year we must see the commonwealth advance in relation to the hygienic and moral control of individuals, not by sumptuary laws or impulsive legislation, but by a true education which will lift the people out of squalor and ignorance and fit citizens for the work of the nation. Thus education will no longer be anchored to ancient formulæ and circumscribed by dead ideals; the process of cramming will be thoroughly and finally discredited; and the individualising of each scholar will be the aim of enlightened teachers.

For the neurotic precocious child ordinary present-day education is a real danger. This has been partially recognised by the establishment of schools for backward children in Leicester, London, and elsewhere (*see* Mental Deficiency); but we have generally failed to control the forward child, whose lively memory and tricks of imitation carry it into the perils of a one-sided development of the nervous system,[1] and often result in disaster. The nervous system is all-pervading, and for the due exercise of every part education must be extended in order to develop every function. Seeing and hearing are necessary to a real assimilation of instruction; yet not only the printed type and the teacher's monitions, but all the avenues of knowledge must be opened up to permit of a general cerebral assimilation. As every muscle is represented in the cerebral cortex, so should every muscle be scientifically exercised. In short, the aim and object of all education must be to secure the sane mind in the sound body.

Hygienic.—That leads, of course, to the consideration of the dangers of unhygienic practices in forms familiar to all. At puberty, when there is danger of the degenerate, endued at conception with a deficient vital force, failing to develop, the precautions above indicated must be redoubled. There must be rest as well as exercise. The brain must not be allowed to rust in idleness,

[1] See article "Hysteria in Childhood," p. 325.

yet mental work must be moderate in intensity and in quantity, and physical excess must be equally regulated. In girls especially at this critical period great caution is required. Fatiguing muscular exertions should be absolutely forbidden, extremes of heat and cold should be avoided. The touchstone of success will be the establishment of physical health, the avoidance of anæmia, the broadening of interests, and the healthy avoidance of evil habits.

In regard to those who have to gain a livelihood comes the question of the choice of a career. That must be considered in view of the mental and physical aptitudes. It seems hardly necessary to advise that neurotic persons should keep as close to mother earth as possible, yet there is a decided inclination among such persons to decide for emotional, exciting, irregular modes of life; and it is difficult to induce them to be content with the even routine of a government office or the bucolic delights of the country, while the stock exchange or the crowded halls of commerce beckon them on with high promises that so often end in disappointment and disaster. "The land for the people" is a cry that comes with convincing appeal in these days of overgrown cities; and when one considers the stunted degenerates of town life, one cannot but re-echo that cry in the interests of the race. The career for neurotic individuals should be free from great responsibilities, should interpose no great difficulties, while it should afford occasion for the exercise of healthy volition. It should be restful in the intervals of work, yet it must be remembered that idleness is not rest—a change of occupation and a variety of interests are both necessary. Kraepelin has shown that mental work added to physical work is provocative of exhaustion; recuperative rest in intervals of brain excitation is not to be gained by bodily exercise. The nervous system is stimulated and exhausted by both these processes, and we have to fall back upon nature's sweet restorer—sleep. We need hardly urge the importance of sound refreshing sleep, or indicate that it must be gained by natural means and not by toxic substances.

That brings us to a brief summary of conclusions as regards the use of alcohol in nervous cases. We have shown that it is a widespread cause of degeneracy, and that it plays a leading part in the production of insanity. We therefore prescribe it with grave unwillingness for the cases under review. Safety for a neurotic individual is only to be maintained by a total avoidance of alcoholic liquors or other drugs of lethe. It may be advisable in rare cases to give it as an anæsthetic in conditions of fatigue and in peril of collapse. It may be required in anæmic complications, but the possibilities of abuse render it a two-edged weapon which we shall do well to avoid using in so far as possible.

There can be no doubt that those of feeble nervous constitution are liable to take extreme views in connection with religion, and guidance is required in this sphere of thought. The green sickness of youth is often characterised by bizarre opinions in religion and politics, which a greater experience of life modifies. Heinroth's theory of the common origin of insanity and sin was fortunately soon put aside by his fellow-countrymen; but every now and then it crops up amongst those afflicted with certain forms of religiosity. The religion of the present day is more concerned with conduct than with forms of profession, and the religion for neurotics is of a moderate, practical nature—avoiding undue introspection or the attempted solution of insoluble questions.

If a person of unstable nervous tendencies decides to marry, the most reasonable advice usually comes too late and is often taken amiss. For the women there are dangers in the probabilities of adverse circumstances and domestic troubles; in too rapid procreation; in the petty worries of life which marriage so often entails. For the men, there are dangers in the greater responsibilities and the social difficulties which they undertake to meet. But objections raised in the consulting-room very rarely carry weight, and until the State interferes to limit the procreation of the unfit, we can only rely upon right education informing the public mind and emphasising the moral code. At present, in this transition stage, it is evident that prudence in marriage is confined to the classes who have least need to exercise it. The criminal, the reckless, the improvident put no bounds to the gratification of their desires, and the breeding of paupers and inefficients is assuredly a danger to the commonweal.

Restrictive measures were not unknown in former days. Howell (*Familiar Letters*, lx. pt. ii.) says—"The countries that are freest from excess in drinking are Spain and Italy; if a woman can prove her husband to have been thrice drunk, by the ancient laws of Spain she may plead a divorce from him."

If we may believe Hector Boece, Scotland did early recognise that the procreation of the unfit should be restrained. He recorded: "If any were troubled with the falling evil, or leprosy, or fallen frantic, or otherwise was out of his wits, they were diligently sought out; and, lest those diseases should pass further by infectious generation into their issue and posterity, they gelded the men. But the women were secluded to some odd place far off from the company of men, where if she afterwards happened to be gotten with child, both she and the infant were run through with the lance. Gluttons and ravenours, drunkards, and egregious devourers of victuals were punished also by death, first being permitted to devour so much as they listed." These were Draconian methods which marked the dawn of civilisation, and there are

yet those who advocate similar measures. But we must limit our proposals to practical suggestions to modify the liberty of the subject to destroy himself and confer an heritage of woe upon his offspring. Legislative protection is not within the sphere of our activities. True, Dr. Toulouse has formnlated a proposal for France, where the consent of parents must be given before marriage, suggesting that the contract of marriage should be protected by an investigation into the physical condition of the parties. He suggests that they should be examined by medical men in the manner incumbent upon those who enter on policies of life insurance. He would declare any marriage void if facts as to morbid heredity were concealed. Certainly such an enactment would direct public attention to the wrongdoing which now proceeds unchecked, but it would bring in its train evils which demand consideration. For instance, how far would the State be justified in branding with illegitimacy the offspring of those to whom marriage was forbidden, thus adding to the burden of their neurotic inheritance social disqualifications which thcy are specially unfit to bear? Our duty lies nearer at hand; we must hope to stem the tide of degeneracy and insanity by our efforts to educate the nation and to guide it in proper directions. Our present restrictive legal enactments fail by reason of their incompleteness, for the inefficacy of the Habitual Drunkards Acts and the Contagious Diseases Acts is deplorable not only in regard to the present generation, but still morc in regard to those who are to come.

Insanity, Pathology of.

A. Pathogenesis

To have the pathology of insanity brought into line with other departments of pathology has long been a desideratum. Most alienists have, however, regarded the task as for the present impossible of accomplishment, whilst not a few have scoffed at the very idea as in the nature of things absurd. In the opinion of the writer, it can be shown that there are certain fundamental principles underlying the development of all disease and applicable no less to mental diseases than to gastric, renal, or hepatic disorders.

The first principle that it is necessary to lay down, is that *every living being is the produce of two developmental factors, namely, heredity and environmental influences.* The term heredity has, to science at least, now lost all significance of transmission from parent to child that it once had. Recent embryological investigation has shown that there is a continuity of germ-cells, and that the individual is the product of one germ-cell, which, at a certain stage in the cycle of the germ-cells, is sacrificed to form a temporary habitation for the rest, which continue their cycle therein. The individual therefore cannot inherit characters from his parents; he is simply the realisation of the developmental potentialities of a primary germ-cell which has descended in direct line from other germ-cells, and which develops in response to the influence of an environment to which there is more or less perfect adaptation. Slight modifications of the developmental potentialities of germ-cells (genetic variation) certainly take place from time to time, and there is conclusive experimental evidence that, in plants at least, they occur in consequence of changes in the environment. In regard to animals, the proof is not so direct, but there can be little doubt that in them also genetic variation is dependent upon similar causes. The conjugation of germ-cells, which takes place at a definite stage in their life-circle, results in the formation of primary germ-cells, the developmental potentialities of which represent the blending of those of the paternal and maternal germ-cells. This blending is, however, influenced by certain laws of exclusive inheritance, which have in recent years been the subject of much investigation, especially in plants, under the denomination of *Mendelism.* In the development of every individual the hereditary factor is fixed; the environmental factor, on the other hand, may vary within very wide limits, and the product will vary accordingly.

The second great principle that it is necessary to insist upon is that *the individual constitutes a vital reactive mechanism.* Heredity and environmental influences build up a highly complex organism, and every vital phenomenon it is capable of exhibiting is a response to external stimuli. This is true of the individual as a whole, of the various organs and of the separate cells composing the tissues. We hear the ring of the telephone bell and we go to the instrument in order to answer the summons; we merely perform a complex reflex action induced by thc representations awakened by the sound of the bell. Similarly, the functional activity of a glandular organ, such as the stomach, is purely a response to stimuli from its environment, which includes the other organs and tissues of the body; and the activity of every

single gland cell is likewise a response to a stimulus from without. This principle applies to the activities of the nervous organs as to those of other organs of the body, and it applies not only to the lower nervous functions, but also to the highest, that is to say, to the mental processes. All the functional activities of the brain, like those of the stomach, liver, salivary glands, etc., are essentially reflexes, or definite responses to stimuli. "In the psychical actions," says Tanzi,[1] "we are obliged always to presuppose the occurrence of antecedent sensorial events which either take origin in the environment, or are cœnesthetic, arising from within; however remote, indistinct, and forgotten this essential antecedent may be, it operates as a physiological stimulus and reduces even voluntary initiative to the level of a reflex, differing from a common reaction only in its greater complexity. . . . Voluntary actions are to be interpreted as intelligent and obligatory responses to the stimuli that continuously impinge upon consciousness in the form of desires; and thus the manifestations of will do not differ objectively from other reflex phenomena, save in their slowness. . . . Voluntary actions blend with instinctive actions, and assume, even when viewed from within, the aspect of reflexes with consciousness, and nothing more."

If we look upon consciousness and conduct as respectively subjective and objective manifestations of complex reactions to stimuli (and there are at least those who are prepared to contend that the data of modern science justify the taking of this view), then mental phenomena can be regarded from the same standpoint as other functional manifestations, and we can proceed to construct a pathology of insanity that is in harmony with general pathology.

Before this end can be fully achieved, however, it is further necessary to establish a fundamental principle of general pathology that is not yet recognised. It is necessary indeed to understand *the biological significance of disease.* In a paper published some years ago, the writer[2] attempted to grapple with this question, and further consideration of the matter has only served to strengthen his conviction that the solution therein offered is in accord with the facts. In the history of the race, genetic variation and natural selection have been continually operative in the direction of producing adaptation to environment. A plant or animal that is perfectly adapted to its environment will pass through life without suffering from disease. All its reactions to environmental stimuli will be purely physiological, and it will ultimately succumb to a simple process of involution. Such conditions are never realised in higher animal forms, owing to the great number and complexity of the external agencies that act upon them. The adaptation is always to a greater or less extent imperfect. In all higher forms of life, any high degree of adaptation to environment implies a state of sustained defence against innumerable inimical forces, chief among which are certain bacterial foes. The great instruments of this protective action are the skin and mucous membranes, which form the individual's first line of defence. There are, however, numerous inimical forces which accident of circumstances may bring into contact with the individual, and against which this first line of defence may be incapable of affording protection. When an inimical force—as for example the bacillus of influenza or the toxins produced by the diphtheria bacillus—penetrates the first line of defence, a second line of defence, constituted mainly by the protective forces that form the mechanism of natural and acquired immunity, is brought into action. There occurs, in other words, a reaction to the inimical force, and such a reaction constitutes disease. We may therefore lay down a third principle, namely, that *disease is a reaction to an inimical force that has penetrated the first line of defence of the organism.* The exact characters of the reaction will vary within wide limits in accordance with the special reactive qualities of the individual. These, like all other reactive qualities, are dependent upon structure, which, as has been maintained, is always the joint product of heredity and environmental influence. It is, moreover, to be borne in mind that in a given individual the bodily structure, and therefore also the reactivity, are continually being modified by environmental influences, and that whilst such modification occurs within only narrow limits in health, it takes place to a very marked and important extent as a consequence of disease. Thus in health it is perceptibly influenced by age, food, fatigue, sleep, etc., whilst it is often profoundly modified by organic changes such as those that occur in a tubercular lung, an atrophied thyroid gland, or a stomach that is the seat of a malignant tumour. It is further to be said in this connection that there is evidence that certain toxæmic conditions induce genetic variation, and although the influence of this may be in the direction of better adaptation to environment on the part of the descendants, it may also be operative in the other direction, and thus tend to increase the incidence of disease.

Disease is thus a consequence of imperfect adaptation. It is a vital process, and is primarily defensive in purpose. It is to be distinguished from traumatism, inanition, and involution, as well as from conditions that are results of disease, such as the morbid tissue changes that may be observed after death.

The functional activities of the brain may be a thousandfold more complex than those of any other organ, but, as far as they can be analysed,

[1] *Trattato delle malattie mentali,* 1904.
[2] *British Journal of Inebriety,* April 1904.

they are still reactions and nothing more. The special reactive qualities of the brain are, as in the case of other organs, dependent upon structure, which in turn is the product of heredity and of environmental influences. The relatively enormous capacity the brain has of being moulded by external physical (sensory) stimuli, and the extraordinary complexity of its reactions, obscure the fact that its structural development and functional activities are dependent upon laws identical with those that control the development and functional activities of other organs.

This simple and incontrovertible fact can hardly be said to have been recognised, and the pathology of insanity, remaining apart from general pathology, has not yet been given either the position or the support that it deserves. Inability to regard mental diseases from the point of view of general pathology has resulted in an unwarranted belief in heredity as a cause of insanity and in extraordinary tardiness in realising the paramount importance of environmental influences as etiological factors. It is still in this country orthodox mental pathology to explain the occurrence of various forms of insanity by saying that the individual has "a bad heredity," or "a neuropathic constitution." Such views err in being wholly unanalytic; they are at the same time unscientific, for they fail to take into account the existing evidence bearing upon the subject, and science is knowledge founded upon evidence; and, moreover, they are constantly associated with an obsolete doctrine of heredity, according to which children are believed to inherit characters from their parents. Heredity alone cannot be a cause of anything; it is only a moulding force which builds a structure out of environmental material. With the same heredity, the nature of the product will differ within very wide limits according to the special and largely accidental characters of the environment. The victim of an attack of insanity, like any other living reactive mechanism, is the joint product of heredity and environment. It may be that certain of his ancestors, the successive tenements of antecedent converging lines of germ-cells, exhibited morbid mental phenomena, but it is impossible to tell how much in these individuals was to be attributed to unfortunate developmental potentialities of the primary germ-cells from which they sprang, and in how far they were the victims of specially unfavourable environmental influences. A history of insanity in ancestors only furnishes presumptive evidence (in many instances quite fallacious) that the original developmental potentialities were such as to favour the formation of an individual with certain special reactive qualities. The environmental factors are so numerous, so uncertain, and so potent that, even if the exact developmental potentialities of a primary germ-cell about to develop were known, it would be impossible to forecast with any exactitude what it would become.

The instinctive actions of man and of lower animals have been aptly described as functional correlatives of structure. The special structures are developed and perfected in a suitable environment without any process of education, and the actions occur as responses to special stimuli. It is also recognised that these instinctive actions tend in course of time to be modified in various ways as a result of experience; they come, in short, to have added to them some of the features of habits. It is not perhaps so generally realised that habits also are functional correlatives of structure and displayed only in response to special stimuli; they differ from instincts merely in the fact that conscious processes have been concerned in their development. The capacity for such modification and development of nervous structure, and consequently for the acquirement of reactive characters higher than those that manifest themselves as instinctive actions, is vastly greater in man than in any of the lower animals. Indeed, it is chiefly in virtue of this greater capacity that he is superior to other animals, and it would appear to be dependent upon a relatively enormous development of associative centres of marvellous impressionability. The more closely their mechanism is investigated, the more clearly does it appear that the functional acquirements of the nervous system are correlatives of structure and manifest themselves only in response to suitable stimuli. Even the very highest mental function, that of will, the further it is analysed the more it is seen to be merely the reaction to a representation of a certain emotional tone. It is true that we can never carry the analysis so far as to get rid of the possibility of there being a final fiat concerned with it; but this cannot alter the fact of the essentially reactive character of mental processes, as well as of the movements originated by them. Mental acquirements are essentially the result of elaboration of sensory stimuli which are passed on from the centres for sensation to centres of representation, or the psychical or associative centres, the existence of which is now a matter of certainty, although there is great difference of opinion regarding their exact localisation. In these centres there is slowly built up a reactive mechanism of marvellous complexity. Consciousness is a concomitant of the occurrence of reaction in these psychical centres. According to Flechsig, whose views are not, however, accepted by all of those competent to form an opinion upon the question, there are three great associative centres, namely—(1) the prefrontal zone, (2) the insular zone, and (3) the parieto-occipito-temporal zone. The centres for speech are included in these zones, their function being psychical in character. It seems probable that

connected with each specific centre of sensation there is an associative zone that forms a corresponding mnemonic repository. Whether this is exactly so or not, "two facts," says Tanzi, "remain beyond dispute, namely, that there are associative stations, and that the anatomical connections by which these stations are united to the known centres of projection are of a specific nature. From the latter fact it is legitimate to infer that differentiated intellectual functions develop in the sphere of these connections." The same authority draws from the ascertained facts regarding the development, arrangement and mode of action of the cerebral nervous elements, some further very striking deductions which may be mentioned here. The psychical processes are always pluricellular phenomena; a single cortical element is not by itself capable of lodging or producing a concrete image, not even that of an image of a luminous point that has impinged upon a single rod or cone of the retina. The cortical cell is polydynamic; every central element (and especially those of the associative centres) has multiple connections with other elements, both peripheral and central, and may therefore be affected by a series of impressions different in origin and to a certain extent also in nature; the function of the cortical cell is therefore very far from being limited to a single form of stereotyped activity, but at the same time it does not consist in an indefinite capacity to receive any kind of stimulus. Every cortical cell is both an organ of reception and an organ of discharge, inasmuch as it passes on to other cells the stimulus it has received. In contact with so many other different kinds of cells, and exposed to so many forms of excitation, the cell of the cerebral cortex acquires the power of reacting in various ways and little by little, through adaptation, even that of not reacting, or of exercising a direct inhibitory influence.

Another very suggestive conclusion is one that has been drawn by Lugaro. He maintains that two distinct elaborations of external impressions occur in the nerve centres, the one inter-neuronic, at the terminations of the afferent fibres, and the other intra-neuronic, between the wave conveyed by the dendrites and the dynamical processes that occur in the interior of the cell-body, and that the first corresponds to the intellectual phenomena, the second to the affective state.

The subjective manifestation of reaction in the associative zones is the stream of consciousness, which has always a certain emotional colouring, and its objective manifestation is included in conduct. The stream of consciousness is initiated and maintained by sensory stimuli, and would quickly cease if these were completely cut off; but it is guided in its course, at least in all educated people who do not follow the mere promptings of instinct, by hierarchical representations or ideals which become habitual reactions, and which constitute the great motive forces of conduct.

As we are observed to react, so we are classified in the catalogue of intelligence as well-informed or ignorant, diligent or lazy, honest or dishonest, clever or stupid, sane or insane. We are here concerned with the explanation of the conditions that determine mental reactions so anomalous as to cause the individual to be placed in the last-named category. It is necessary to remind the reader, however, that insanity is essentially a legal distinction. Moreover, if the pathology of insanity is to be brought into line with general pathology, we must begin to distinguish between "mental disease" and "insanity," for the former consists in internal reactive processes, the latter is marked by the occurrence of anomalous functional reactions. In other words, mental diseases (on the analogy of gastric diseases and renal diseases) are reactions to an inimical force that threatens the integrity of those nervous tissues that subserve the intellectual functions; insanity, on the other hand, essentially implies, as we shall see, the presence of a reactive mechanism that is defective, as a result either of disease or of genetic or germinal variation. Biologically it is a lack of adaptation to environment. Practically it is a particular form of racial degeneration or defectiveness, about which so much has been said in recent years.

How, then, are those anomalous functional reactions that we call insanity brought about? The possible factors are very numerous and require to be carefully analysed and distinguished. It is, however, impossible to enter into the subject in any detail here, and therefore only what appear to the writer to be the important principles will be sketched. Broadly speaking, either the external stimulus or the reactive mechanism must be at fault.

The influence of abnormal sensory stimuli is greater than might at first appear. Here, however, it is essential that there should be no mistake as to the point of view from which the vital processes are being regarded. The reactive mechanisms with which we are concerned are those that immediately subserve the mental functions, that is to say, the associative centres. Other portions of the nervous system, including the peripheral and central sensory organs, are external to these centres and the stimuli that they convey to them are external stimuli. Among external stimuli of this kind are to be included fright, the receipt of bad news, and other similar causes of severe emotional disturbance. It may be questioned if such influences are capable of causing insanity without the previous establishment of some defect of the central reactive mechanism, but that they occasionally do determine the occurrence of insanity is certain. Their action may be so

severe as to produce shock, and in this case is equivalent to traumatism. When insanity arises as a more remote consequence it is dependent upon a general toxæmia induced by emotional disturbance, which causes disorder of the functional action of various non-nervous organs. Insanity arising from excessive stimulation of the associative centres, or mental over-strain, is also essentially dependent upon general toxæmia similarly induced. The evidence of experimental physiology goes to show that nerve tissues are inexhaustible, and that therefore the action of over-exertion in producing insanity is not a direct one. It is probable that in most cases in which nervous over-strain and affective disturbances are followed by insanity, some other toxic condition, or some defect in the reactive mechanism, is already present, without which the cause to which the insanity is usually attributed would not have produced it. Simple sensory disturbances, if very intense or unusual, such as severe neuralgia affecting the fifth nerve and the irritation produced by intestinal worms, may occasionally cause psychopathic disturbances. Lesions of the special sense organs, by rendering correspondence with the environment defective, may cause insanity. As a rule, however, a compensatory action on the part of other sense organs is displayed and the effects of the lesion are more or less completely nullified. A special lesion of the sensory centres is, according to the hypothesis of Tanzi, which seems to be in agreement with the available evidence, the cause of most forms of true hallucination. According to this hypothesis, an hallucination has its origin in a representational centre from which a stimulus (and idea or symbol) passes back to the sensorial centre, where it assumes the aspect of a sensation and is mistaken for a real sensation. The fault consists in a morbid permeability to stimuli on the part of certain afferent fibres which normally probably control the process of attention. Disturbances or lesions affecting the somæsthetic centres may seriously modify the emotional tone, giving rise in some instances to euphoria, in others to severe mental depression.

In the production of the mental reactions, ideational stimuli are of paramount importance, and therefore it might seem that abnormal ideational stimuli should be included here; but such ideational stimuli are themselves evidence of reaction induced by other stimuli, and the reaction is one that takes place in the psychical centres. Abnormal ideational stimuli are therefore dependent upon defect of the reactive mechanism.

It must be evident that abnormal external stimuli, acting apart from other morbid conditions, can have only an insignificant share in the causation of insanity. In the vast majority of cases it is therefore the reactive mechanism that is at fault. We have then to inquire in what ways the reactive mechanism constituted by the associative centres may become so abnormal as to determine insanity. As in the case of all other organs the psychical centres are the product of heredity and environment, and as their structure varies so will their reactivity vary.

There is first the possibility of the occurrence of genetic variation of such a nature as to result in the development of associative centres so anomalous that their reactive manifestations are regarded as insane actions. Such an occurrence is probably not so common as is sometimes supposed, for it is certainly closely simulated in its effects by another essentially different condition, namely, unsuitability of the blending of two ancestral lines, intensified often by the operation of the law of exclusive inheritance. In compensation for such occasional disastrous results of genetic variation or of germinal variation, there are converse cases in which either genetic variation or the fortunate combination of two ancestral lines produces an individual of far above ordinary capacity for intellectual development. We are concerned here, however, only with the unfortunate deviations of these kinds from the normal lines; they probably account for most cases of paranoia, certain forms of idiocy and imbecility, some cases of moral insanity, some of the obsessive psychoses and certain forms of hysteria. Genetic variation may also affect non-nervous organs, which consequently manifest imperfect adaptation to environment, and so give rise to disorders of metabolism which are responsible for the formation of toxic substances which may injure the nervous tissues and so affect their reactivity. There seem now to be grounds for the belief that proneness to the development of certain forms of insanity depends not upon any inherent cerebral defect as is so commonly assumed, but upon a special tendency to the development of certain disorders of metabolism or to chronic infections by organisms that are ordinarily only saprophytic. In this category there are probably to be placed the whole group of the affective psychoses and also dementia præcox.

In the building up of the associative reactive mechanism, the powerful influence exercised by education must not be forgotten. Defective or vicious education, to be included in which is the moral influence of a bad environment such as that which is to be found in the slums of our large cities, is probably to an important extent responsible for cases of alcoholism, criminality, and moral insanity.

Traumatism and its consequences occupy a considerable place as a cause of morbid alteration in the associative reactive mechanism. Head traumatism occurring at birth is probably a far more common cause of imbecility and epilepsy than is generally believed. Similar injuries occurring at later periods of life, as well

as the shock of severe accidents, give rise to a large variety of traumatic psychosis.

The development of the great majority of cases of insanity has not yet been accounted for, and there remains only one important condition to which they can be attributed, namely, toxic action. If the cerebral functions have developed in a normal manner, and if afterwards the person suffers from an attack of insanity, there are, with the rarest exceptions, really only two conditions that can explain the departure from the normal, namely, traumatism and toxic action, and the part played by the former is certainly a comparatively small one. It is true that inanition and involution have not yet been considered, but when these are analysed they both ultimately resolve themselves into toxæmias. Objection has sometimes been taken to attaching great importance to toxæmia in the etiology of insanity because of the fact that the metabolic changes occurring in the nervous tissues are extremely small in amount. This objection to the toxic etiology of insanity is, however, rendered untenable by the fact that the metabolic changes that do take place are of great importance for the elements concerned. "The association of a very small amount of chemical change with the exquisitely fine chemical sensibility is what constitutes the dominant characters of nervous metabolism. The brain serves for the redistribution, not for the production of energy; it has been fittingly compared by Belmondo to a commutator" (Tanzi). The sensitiveness of the cortical nerve-cells to toxins is shown by various facts of common experience. Ordinary fatigue is due to the accumulation of physiological toxins and the brain is quickly influenced by them. In most illnesses of toxic origin, general lassitude and disinclination to exercise the mental functions are among the most constant symptoms. Various drugs, such as morphia, cocaine, strychnine, etc., influence the cerebral nerve-cells with extreme readiness. Moreover, there is ample experimental evidence that nerve-cells are easily damaged by certain toxic agents. It is therefore futile to attempt to belittle the importance of toxic action as a cause of insanity on the ground of the small amount of metabolic change that occurs in the nerve-cells.

Attempts have also been made to restrict the meaning of the term toxin in a way that serves only to make false distinctions and to create confusion. It is useless to limit the meaning of the term, when disease is being regarded from its biological standpoint as a reaction on the part of the cells of the body to an inimical force to which they are attempting to adapt themselves (the process of somatic variation). Any chemical agent to which a cell is not adapted (unable to utilise or to protect itself from) may act as a toxin to that cell.

The chief forms of toxic action concerned with the production of insanity may be summarised as those resulting from simple intoxication or poisoning, infections and auto-intoxications. Simple intoxications include those arising from the taking of alcohol, morphia, cocaine, lead salts, damaged maize, etc. Infections include those associated with typhoid fever, pneumonia, meningitis, influenza, malarial fever, puerperal fever, syphilis (second stage), each of which may be complicated or followed by insanity, and also general paralysis of the insane, which, according to the researches of Dr. Douglas M'Rae and the writer, is dependent upon infection by one or other of two varieties of diphtheroid bacillus of special virulence. All of these infective conditions, through the action either of the primary toxins or of secondary ones, may so alter the functional reactivity of the cortical nerve-cells as to produce the amential syndrome, characterised especially by mental confusion. This syndrome would appear to be the characteristic clinical manifestation of widespread toxic processes actually taking place in the nerve-cells of the associative centres. Its extreme form is acute delirium.

Another important form of infection is chronic toxic infection by micro-organisms that are ordinarily only saphrophytic, which arises in consequence of injury to the first line of defence, more particularly in the alimentary tract. This condition occurs especially in chronic alcoholism, general paralysis, senile insanity, pellagra, the uremic psychoses, and in chronic constipation.

Auto-intoxication is in the majority of instances consequent upon some previous pathological process which has impaired or destroyed the functional activity of certain organs, and has rendered the individual in certain respects unadapted to his environment. This occurs, for example, in cretinism and myxœdema in which the atrophy of the thyroid gland can only be accounted for as a consequence of a morbid process and not as a genetic variation. A similar explanation holds good for those forms of auto-intoxication that depend upon impairment of the hepatic, renal, adrenal, pancreatic, and parathyroid functions, as well for the auto-intoxications that occur in exophthalmic goitre and diabetes mellitus. As already indicated, some cases of auto-intoxication are also to be attributed to lack of adaptation resulting from genetic variation and to affective disturbances of psychical origin.

The changes produced in the cortical nerve-cells by toxic action may be recovered from, or they may be permanent. When they are permanent, there is some corresponding permanent change in the functional reactivity. A typical example is seen in secondary dementia. There are, however, many other morbid conditions that are to be similarly explained as dependent upon an irreparable lesion affecting the associa-

tive reactive mechanism, such as simple loss of mnemonic records, psychical blindness, aphasias, certain obsessive ideas, pseudo-hallucinations, and permanent changes in character.

Reference must also be made to the occurrence of toxic lesions in the nervous centres during intra-uterine life. These may be of all degrees of severity; their consequences may be early manifested in the form of infantile cerebropathies accompanied by idiocy, imbecility, and often epilepsy. It is probable that many other manifestations of defective cerebral development have a similar origin, including most cases of epilepsy that arise in adult life.

Cerebral vascular lesions and the gross lesions that result from them are essentially toxæmic in origin; changes in the neuroglia and membranes of the brain are chiefly secondary to toxic conditions that have affected the nervous tissues.

When the factors and processes concerned in the development of insanity are examined from the standpoint of general pathology, it becomes evident that the inimical forces against which the individual is constantly or occasionally called upon to defend himself are by far the most important causes of insanity, and that among these forces toxic, and especially infective, agents play the largest rôle. They produce insanity by altering the structure of the reactive mechanism that constitutes the physical basis of mind. That one person more readily becomes insane than another under similar conditions, is due partly to differences in the cerebral tissues, but probably in still greater measure to differences that concern the whole organisation of the individual.

It will be seen that under the view of the pathology of insanity that is here taken we are concerned not only with the brain as a reactive mechanism, but also within the internal and external stimuli to which it reacts. Lastly, it may be added that in the various forms of insanity, as in other kinds of disease, there are, from the standpoint of pathology, three possible objectives of treatment, namely—(1) to suspend or arrest the action of inimical forces; (2) to restore, as far as may be possible, to a normal condition reactive mechanisms damaged by pathological processes or originally anomalous; and (3) to adapt the environment to defective reactive mechanisms in cases in which these cannot be restored to a normal condition.

B. Pathological Anatomy

The Scalp.—Morbid adhesion of the epicranial aponeurosis to the pericranium is present in about 50 per cent of persons dying insane. An extreme degree of this adhesion, such as to render detachment of the scalp impossible except by dissection, occurs in over 10 per cent of cases. The condition is most common in general paralysis and in epilepsy. In a large majority of instances it is associated with either thickening or condensation of the bones of the skull.

"Hypertrophy of the scalp" is a rare abnormality which appears to have been observed, at least in a well-marked form, only in the mentally defective, and in the majority of instances in genetous idiots. It consists in an abnormally redundant, thick, lax, and mobile condition of the integument of the upper and posterior parts of the head, which at the same time presents a number of roughly symmetrical, generally antero-posterior and transverse furrows and ridges.

Hæmatoma auris, which is exceedingly common in the insane and comparatively very rare in other persons, is described in vol. iv. p. 18.

The Skull.—Morbid conditions of the skull consist of two distinct classes, namely (1) variations in form and size overstepping the limits of the typical or normal, and (2) changes in the structure of the bone, evidenced by abnormalities in thickness, consistence, and histological characters.

The skulls of the insane in this country have not yet been sufficiently studied by strict craniological methods to allow of precise statements being made regarding the special morphological features that they exhibit. Only a few of the more distinct abnormalities that have been observed need be mentioned here.

The microcephalic skull, which exhibits a characteristic form as well as an abnormally small size, has, according to the observations of Sir George Humphry,[1] an average circumference of 16·7 inches. This authority has described its chief characters as follows:—"The brain-case is small or contracted in all its dimensions, in the base as well, though less than in the vault; and it is most so in the fore part, the frontal bone being sloped back and narrow, with deep temporal fossæ behind the external angular processes, and with curved orbital plates, which narrow the ethmoidal fossæ. The parts in the interior are contracted and often thickened."

The macrocephalic or hydrocephalic skull has its greatest circumference above the glabella and occipital point. The authority just quoted has described its chief characters as follows:—"The brain-case is expanded in all directions, to some extent in the base, but much less than in the vault. The forehead bulges forwards, beetling over the face; the orbital plates are in the young flattened and pressed down, and the superciliary ridges are elevated and depressed. The temporal fossæ are shallowed, and the occipital condyles are not prominent."[2]

Although asymmetry of the cranium is not necessarily an indication of an abnormal brain, high degrees and certain special forms of it are

[1] *Journ. Anat. and Physiol.* vol. xxix. 1895, p. 307.

[2] *Journ. Anat. and Physiol.*, *loc. cit.*

certainly far more common in the insane than in the mentally sound.

A special type of deformity, to be observed in some cases of idiocy and imbecility, has been termed *crania progenea*. It consists in abnormal prominence of the lower jaw and forehead, defective development of the occipital region, and narrowing of the face.

Another abnormal form of cranium has been termed by Clapham and Clarke "the insane type," having been found only in cases of insanity. Its special feature consists in the greatest transverse diameter being in its anterior third instead of behind the middle point.

Absence of the normal overriding of the dentary arcades, which the researches of Sir William Turner[1] have shown to be an atavistic character, has been found to be much more common in the insane than in the mentally sound.

Most authorities upon mental diseases at the present day regard deformities of the hard palate as an important sign of a bad initial neurotic heredity. The accuracy of this view is, however, strongly contested by certain other authorities upon such subjects, and therefore remains in question.

Alterations in the thickness, texture, and weight of the cranial bones are exceedingly common in the insane. The calvarium is generally affected to a much greater extent than the base. Distinct thickening may be observed in about 50 per cent of cases examined after death. The change may be local or general, and may be combined either with osteosclerosis or osteoporosis. It is most common in cases of general paralysis and senile insanity. Atrophic thinning of the skull also occurs, but is comparatively rare. Most of the examples are found in cases of senile insanity. Condensation of the bones of the skull, or osteosclerosis, is present in about 30 per cent of all cases. The condition is practically a constant one in general paralysis. It is accompanied by thickening in the majority of instances. General softening of the bones of the skull, or osteoporosis, has been observed to occur in about 5 per cent of cases.

The cause of these alterations in the texture of the bones of the skull in cases of mental disease is probably the same as that which leads to the changes in the dura mater presently to be described.

The Dura Mater.—Morbid adhesion of the dura to the skull is common in various forms of insanity. It has been found by different observers in from 15 to 44 per cent of cases. The condition is probably in most instances related to the circumstance that the periosteal surface of the membrane is depositing new bone. Morbid adhesion to the pia arachnoid is comparatively rare, and is almost always of small extent. It is probably of inflammatory origin. General thickening of the dura is very common in the insane, but is somewhat difficult to recognise with the unaided eye. It depends upon the occurrence of a slow sclerotic process, the evidence of which can be readily observed on microscopic examination. Atrophic thinning of the membrane is very occasionally met with, mostly in cases of senile insanity. Small bony nodules are sometimes present, either in the substance of the dura or on its inner aspect, most commonly adhering to the falx. In most instances they probably represent merely a developmental anomaly in a membrane of which one surface is a periosteum. Inflammation of the dura mater has little or no special relationship to insanity. The so-called *pachymeningitis hæmorrhagica interna* can be shown to be of different origin. On the other hand, degenerative changes are exceedingly common, and have considerable pathological importance. Subdural false membranes can be recognised to be present in about one-fourth of cases examined post-mortem. They vary greatly in size, appearance, and consistence in different instances. They may be limited to a small portion of the dura, or extend over the entire surface of the cranial and spinal membranes. When they are large, and at the same time somewhat localised, they are generally cystic in the centre. They are always more or less adherent to the dura, almost never to the pia arachnoid. Their pathology has given rise to a long-standing controversy, which has been chiefly concerned with two opposing theories regarding their origin, namely, the inflammatory and the hæmorrhagic. According to the writer's observations two classes of subdural false membranes require to be distinguished. One is the result of a simple hæmorrhage into the subdural space, most commonly from a pial vein. The other depends upon certain degenerative conditions of the dura, which are exceedingly common in cases of chronic insanity, more especially general paralysis and senile insanity. In order that we may be able to appreciate fully the nature and importance of these degenerative changes, it is necessary to understand that the dura mater contains innumerable minute lymphatic channels which pass from its inner surface and discharge into the veins both in the membrane and in the cranial bones. These channels convey away the cerebro-spinal fluid. At the inner aspect of the membrane, immediately underneath the surface endothelium, there are very numerous large capillaries, each of which lies in one of these lymphatic channels, which in this position are generally referred to as the perivascular canals of the dura. The walls of these channels are nourished chiefly by the cerebro-spinal fluid which flows through them. In many forms of mental disease this fluid has distinctly toxic properties, owing mainly to the disordered metabolic changes going on in the cerebral

[1] *Journ. Anat. and Physiol.*, 1891, July.

tissues, but partly also to general toxic conditions. Hence the endothelial cells of the dural perivascular canals are irritated and imperfectly nourished. They consequently undergo proliferative and degenerative alterations, which result in compression and obliteration of the superficial capillaries. At the same time general proliferation of the dural tissues tends to occur. Obliteration of the original superficial capillaries is constantly followed by the throwing out of new capillaries on the surface of the membrane. These new vessels tend to give way under various circumstances, but especially during the last few days of life in all cases in which the patient sinks slowly. The great majority of the subdural false membranes that are to be observed in the insane are developed as the result of hæmorrhages brought about in this way. With few exceptions they have the histological characters of recent formations. They are essentially composed of blood-clot, which, however, is commonly in course of being vascularised. Associated with these pathological processes there are various other proliferative and degenerative changes in the tissues of the dura mater leading to the formation of surface granulations, hyaline concentric bodies, mulberry bodies, etc.

The Pia Arachnoid.—The leptomeninges are composed of white fibrous tissue, or perhaps of a special form of elastic tissue, which is arranged in such a way as to form a spongy lymph sac. This structural arrangement extends throughout the whole of the membrane, which, upon this and other grounds, Dr. James Middlemass and the writer have contended, contrary to the general teaching, should be regarded as a single structure. It is important to understand that the pia arachnoid is not directly nourished by the vessels it contains. These are simply passing to and from the cerebral tissues. The membrane depends for its nutrition upon the lymph or cerebro-spinal fluid which, escaping from the capillaries of the brain, passes along the adventitial lymph-channels of the intracerebral vessels, enters the arachnoid spaces, and passes across the subdural space into the perivascular canals of the dura mater.

The most important pathological alteration that affects the pia arachnoid in cases of insanity is one which presents itself as milkiness, thickening, opacity, and other related abnormal appearances. Over 70 per cent of cases of insanity show more or less pronounced evidence of these changes on post-mortem examination. The morbid process is essentially the same as that which occurs on the opposite side of the subdural space, and has the same cause, but differs in respect of the fact that it is not complicated by the vascular changes which occur in the dura. The endothelial cells lining the arachnoid spaces and the outer surface of the membrane undergo proliferative and degenerative changes. At the same time a slow hyperplasia of the connective tissues takes place. Hence the membrane increases in thickness and assumes a milky appearance.

The Cerebral Blood-vessels.—These present several special anatomical features which it is necessary to understand in connection with the study of the pathological changes to which they are subject. All the blood that they convey has to pass (with one or two immaterial exceptions) through the nervous substance. The pia arachnoid has no capillary circulation of its own, and hence there is no possibility of short-cutting of the cerebral blood-supply. The small pial arterioles anastomose somewhat freely with each other. The cerebral capillaries are peculiar in that they possess an external coat. This is composed of highly elastic connective tissue fibrils continuous with those of the adventitia of the arterioles and venules. A special set of capillaries, coming off from the pial arterioles, supplies the first layer of the cortex. All the intra-cerebral vessels have normally in their walls a small amount of a pale yellow substance which increases with age.

One of the most important morbid changes to which the cerebral vessels are subject is the hyaline fibroid degeneration of Gull and Sutton. It consists in hyaline swelling, or of fibroid thickening, of the connective-tissue fibrils of the adventitia. The first represents the degenerative and the second the reparative phase of the morbid process. This form of disease occurs in arterioles, capillaries, and venules. It specially tends to affect the capillaries of the first layer of the cortex, which in senile insanity are often extensively obliterated by it. Another exceedingly important vascular change in cases of chronic insanity is endarteritis deformans, or atheroma. It affects not only the large cerebral arteries, but also the small arterioles and capillaries within the brain. It leads to narrowing of the vascular channels, and not infrequently to their obliteration. Both this vascular alteration and hyaline fibroid degeneration lead to the formation of atrophic softenings. The pathology of endarteritis deformans is still in dispute, but, on the evidence of the changes that may be observed in the cerebral vessels, the disease appears to depend upon an abnormal condition of the blood, in consequence of which the tissues composing the intima are injuriously affected in their nutrition, and undergo either proliferative or degenerative changes. With the temporary cessation of the nutritive disturbance reparative processes occur, resulting always in some increase in the thickness of the intima. The degenerative phase is constantly accompanied by weakening of the muscular coat of the arterioles, and, if associated with high blood-pressure, is apt to lead to local dilatation and the formation of miliary aneurysms. In correspondence with the special frequency with

which their cerebral arterioles are affected by atheroma, the insane are specially prone to the occurrence of cerebral hæmorrhage, which is connected in most instances with the rupture of a miliary aneurysm, or of an arteriole otherwise weakened by this form of morbid change. Endarteritis obliterans affects both the large and the small cerebral arterioles in the vascular form of syphilitic insanity. Acute periarteritis of the intra-cerebral arterioles is an almost constant change in the advanced stages of general paralysis. Colloid degeneration and calcification have occasionally been observed in cases of insanity, but are certainly rare.

The Neuroglia and Mesoglia.—The question of the normal structure of the neuroglia has given rise to much discussion. The tissues to which this name is commonly applied consist really of two totally different cellular elements, the one of which is epiblastic in origin and the other mesoblastic. The evidence in support of this view consists mainly (1) in the fact of the differentiation of the latter tissue-element by means of the platinum method; and (2) in its harmony with the important observations of Capobianco and Fragnito upon the histogenesis of the neuroglia. The true neuroglia consists of cells with numerous, only very slightly branched, specially differentiated processes, at least many of which are attached to the vessels and other structures. The mesoglia in its typical form consists of cells with numerous processes, which branch dichotomously, and are never attached to the vessels or to other structures. The two kinds of cell can be shown to behave quite differently in pathological conditions. The neuroglia serves to support the nervous elements, and it is the tissue of repair in the brain. There is conclusive evidence that the mesoglia cells do not subserve either of these functions. Their rôle seems to correspond pretty closely to that of connective-tissue corpuscles in other organs. It is at least certain that they are capable of acting as phagocytes.

In conditions of chronic irritation in the brain, as in general paralysis, choreic insanity, and chronic alcoholism, there is usually well-marked hypertrophy and proliferation of the neuroglia in certain parts of the brain. Similar changes occur wherever the nervous tissues have been extensively destroyed, as for example in the vicinity of the atrophic softenings so common in cases of senile insanity. The mesoglia cells form a certain proportion of the granular corpuscles (others being leucocytes) which commonly occur wherever dead cerebral tissues are being absorbed. In some irritative conditions they tend to proliferate and to degenerate into amyloid bodies.

The Cortical Nerve-cells.—In recent years very remarkable advances have been made in our knowledge of the structure of the nerve-cell. Two chief classes of nerve-cells are distinguished, namely the *somatochrome*, which contain chromophile substance in their protoplasm, and the *karyochrome*, which are generally much smaller and do not contain this substance in their protoplasm. The somatochrome nerve-cells are by far the more important, and they alone need be dealt with here. The nucleus possesses a distinct limiting membrane, a reticulum and granules, and a single nucleolus. All of its structural elements are acidophile in reaction, with the exception of three or four small particles adherent to the nucleolus, which are basophile, and which constitute the whole of the chromatin in the cell. The protoplasm surrounds the nucleus, and extends on the one hand in the form of long-branching and gradually tapering processes, termed the protoplasmic processes or dendrites, which at some little distance from the cell-body are clothed with minute, often filiform, appendages, the gemmulæ or thorns; on the other hand it extends in the form of a single process, termed the axis-cylinder process or axon, which at a short distance from the cell-body generally gives off some delicate branches, the collaterals. The main process may pass into a medullated nerve-fibre, or run a comparatively short course as a non-medullated fibre. The dendrons are organs of reception of the nervous impulses, the axon the apparatus concerned with their discharge. The protoplasm contains innumerable minute fibrils, which are now generally regarded as the conducting element of the nervous impulses. These fibrils course for the most part independently in the processes, but form a network in the cell-body. The interstices of this network contain localised aggregations of a substance which presents a strong affinity for certain dyes, such as toluidin blue. These aggregations constitute the Nissl-bodies or chromophile particles of the nerve-cell protoplasm. There is generally a collection of a pale yellow substance, the nerve-cell pigment, in the vicinity of the nucleus. Several minute lymph canaliculi course within the body of the larger cells and open upon the surface. They were first observed by Golgi, who described them as a special endocellular reticulum.

The neuron theory, according to which each nerve-cell (with its processes) is anatomically and physiologically an independent element, has lately been boldly attacked by Apáthy, Bethe, and Nissl, but, nevertheless, retains its place as a canon of neurology. It is still defended and maintained by such high authorities as Lugaro, Marinesco, van Gehuchten, Ramon y Cajal, Barker, and Lenhossék.

It has now been definitely proved that the chromophile substance of the protoplasm is utilised during the functional activity of the nerve-cells.

Recent experimental investigations have thrown a great amount of light upon the mean-

ing of many of the abnormal appearances to be observed in the nerve-cells of the human subject. It has been shown that the nerve-cell is exceedingly sensitive to the action of many different toxic substances, and that it undergoes definite structural alterations after injury to its axon. The pathological process set up by the former cause has been designated *primary degeneration*, and that resulting from the action of the latter *secondary degeneration*. In primary degeneration the changes vary considerably in accordance with the toxin employed and the intensity of its action. In general the condition manifests itself first by disintegration of the chromophile substance of the protoplasm (*chromatolysis*), and afterwards by disintegration of the fibrils, degeneration of the nucleus, and death of the whole element. If the alteration does not proceed further than simple chromatolysis, it may be recovered from. In secondary degeneration the alteration begins with disintegration of the chromophile substance in the neighbourhood of the cone of origin of the axon, and gradually extends to the rest of the cell-body. At an advanced stage the nucleus is often displaced to the periphery, and the primitive fibrils of the protoplasm destroyed. The cell may undergo repair, or it may atrophy and disappear.

Primary and secondary degeneration are the only diseases of the fully developed nerve-cell that are at present definitely known. Further research will doubtless enable us to recognise various pathologically distinct varieties of primary degeneration. The many types of morbid change that may be observed to affect the nerve-cells of the human cerebral cortex are to be regarded as indicative of the occurrence of one or other of these two forms of degeneration, and must not be looked upon as in themselves distinct diseases of the cell. Among these types of morbid change the most common are chromatolysis, pigmentary or yellow globular degeneration, vacuolation, varicose atrophy of the protoplasmic processes, varicose hypertrophy of the axis-cylinder process, and displacement of the nucleus.

The following are some of the more important facts that seem now to have been definitely established regarding the pathological alterations in the cortical nerve-cells in different forms of mental disease. In cases of acute mania and acute melancholia dying in the course of the disease, from 25 to 50 per cent of the cells show distinctly marked degeneration of the primary type. This percentage is very much higher than that which is generally to be found in persons succumbing, for example, simply to pneumonia, or exhaustion from any wasting disease, such as phthisis. Moreover, the intensity of the pathological changes is far greater in cases of acute insanity, and there is satisfactory evidence that many of the cells have completely disintegrated and disappeared. In senile insanity and in general paralysis there is constantly a slow degeneration of the cortical nerve-cells, for the most part of a primary type. The number of cells involved is very much larger than that affected in normal senility, and the severity of the alterations is far greater. In general paralysis considerable areas of the cortex are often practically depleted of nerve-cells. In cases of choreic insanity there are always extensive, more or less acute, degenerative changes in the cortical nerve-cells, also of a far more severe character than those that occur from simple terminal auto-intoxications. In chronic alcoholic insanity there is very considerable loss of cortical nerve-cells, while a large proportion of those that remain show degenerative changes either of a primary or of a secondary type. Secondary dementia depends essentially upon the fact that a large proportion of the cortical nerve-cells have suffered complete degeneration. In some cases it can be shown that about 50 per cent of them have entirely disappeared.

Congenital Abnormalities of the Brain.—Some of the slighter of these may not infrequently be observed in persons who, during life, did not exhibit any noteworthy mental aberration or peculiarity; nevertheless such slight structural abnormalities (such as atypical arrangement of the convolutions and sulci, absence or imperfect development of various parts of the brain, heterotopia of the grey substance) are far more common in the insane than in the mentally sound. They are especially common in cases of idiocy, imbecility, and epileptic insanity. Some of the more gross congenital defects, such as microcephaly, are quite incompatible with normal intellectual development, while others, such as porencephaly and absence of the corpus callosum, have only very rarely been discovered in sane persons.

Focal Lesions.—Localised gross lesions of the brain are present in a remarkably high percentage of cases of insanity examined after death. The form and degree of mental derangement that they occasion probably depend mainly upon their localisation and the special reactive qualities of the individual brain. In a large majority of instances they are not, however, the only important structural alterations in the organ, being associated with well-marked pathological changes in various other situations.

By far the most common and most important focal lesions are those of vascular origin. They are represented especially by atrophic and hæmorrhagic softenings. The former especially tend to be multiple. Both are extremely common in the senile insane, but are also not infrequently to be observed in lunatics dying in middle life.

Various forms of cerebral tumour are common

exciting causes of insanity. Tubercular nodules are also to be observed with some frequency in the cerebral substance. Localised gummata are occasionally present in cases of syphilitic insanity, but appear to be less common than a diffuse gummatous infiltration of the pia arachnoid and adjacent brain substance, which is generally associated with endarteritis obliterans of the cerebral arteries. Extensive localised areas of sclerosis are occasionally met with, especially, but not exclusively, in epileptics. Their origin is still somewhat obscure. A special form of localised sclerosis of the brain, which, like the preceding, occurs especially in epileptic idiots and imbeciles, has been termed *hypertrophic nodular gliosis.* The nodules vary in size in different instances. They are generally multiple and affect especially the cortex. According to the observations of Pellizzi,[1] this form of cerebral sclerosis depends upon a defect of development arising at a time corresponding to the formation of the tertiary sulci.

Insanity, its Nature and Symptoms.

[1] *Annali di freniatria*, 1899, f. 4; 1900, f. 1, 2.

I. The Relation of Mind and Brain

(1) Biology of Consciousness.—The medical faculty, as well as the general public, still labour to some extent under a time-honoured disability in their view of insanity, for which our philosophers are largely to blame. To the philosopher, indifferent to the pedigree of brains, and habitually averse to appreciate the material conditions of thought, the development of personality is interpreted merely as an evolution of consciousness, sometimes consecutive and intelligible, but often inconsequent and fortuitous. For the phases of the human mind, subjectively considered, resolve themselves into moments of self-attention, alternating with periods of attention to things other than the self, and without any apparent thread to bind them together. The attention moves from this to that, sometimes by association of ideas, sometimes because of a fact in the environment which it perceives; but very often also without any apparent occasion either in itself or in its circumstance. Every human mind has gaps in it. Alone, in still darkness, things jump into the mind; and, at any moment in which the attention is not following an idea, nor noticing the outer world, irrelevant suggestions occur. Such interruptions or invasions of the mind are only to be explained by a view of personality which relates it to cerebral processes, some of which are determined by self-directed movements of the attention, some as reactions to the experience of the moment, but also some which are automatic. And especially when we consider insanities and allied states we must have recourse to a cerebral interpretation of the facts. Most people still labour under a view of insanity which regards it merely as the lost wanderings, or the transcendent flights, of the soul. That was an orthodox view about a century ago. Insanity is cerebration—the activities of a disordered cortex—and the modes of consciousness which it exhibits are to be explained as due to realms and tracts whose functions are normally subconscious. It must become our habit to think of the mind as conditioned by nervous states, and of personality as determined by inherited cerebral character. Only by an explicit and detailed study of the mutual relations of spirit and brain can we appreciate either normal personality or these interruptions or invasions of it—loss of power in memory, in judgment, or in purpose, occasional lapses to brute states, muscular violence, emotional frenzy—to which the unstable are liable.

The first step towards an appreciation of the cerebral relation of mind is a biological one. We must conceive of our brain-stuff as not only the vehicle of all that is forthcoming in human character, but also as preserving more or less active rudiments corresponding to the unrecognised memories of all animal and other ancestral experience. The human mind is at once the master and the servant of an organ of whose potencies the sane and orderly person realises only a very small fraction, but an organ which, in dissolution, reveals an infinite store of atavistic associations—"instinctive vestiges," as Professor Hall calls them. Of these, the more distant, the prehistoric, come only slightly if at all to the surface; but insanity teems with reminiscences of a more recent stage—a stage which corresponds to the somewhat rapid transition from uncivilisation into polite society. Of all that history a review were well worthy of our consideration, a revision of the salient points in comparative physiology which denote the several developments of animal consciousness, as one function after another became for the moment the prime activity in the life of the species. However elaborate we may make our study of the histology of the present-day brain, we cannot by that means understand much of its significance, but only its mass and its complexity. It is only by recalling how, in previous forms, so far as we know, the consciousness embraced one function after another, and how each was superseded by another, but not entirely lost, that we can appreciate the multitude of important relations which lie buried, and not very deeply, beneath our present conscious level. And as we glance at a few of the most important phases of consciousness in its evolution, we should note all through a few general facts whose importance may become apparent later on. In particular, let us remember that character is stigmatised not only according to the things which the mind attends to, but also by its mode of regarding them. Two minds may embrace the same things in their experience, but their embrace of them may be entirely diverse. Insanity reveals an alteration from the normal consciousness in both respects. In insanity there is always under-attention to some things and over-attention to others. But the mode as well as the range of attention may be insane. Insanity is a failure of the normal personality, whereby the mind is reduced to a lower level of consciousness. And the modes of consciousness which it reveals are vestiges of a previous stage of our evolution. For example, perhaps the most important mode of insane consciousness, the fugitive, is one to which we shall often refer. It is a mode of mind which is afraid, has the sense of being hunted or persecuted, is suspicious, unfriendly, and querulous. That is obviously a reversion to a kind of mind which was normal to a stage of evolution in which life was much more dangerous and much less kindly than now. But it is merely a sample of many such vestiges.

Our study resolves itself into an account of the human mind as the physician ought to know it, whether in health or in disease. To him, mind is an evolution which has come by very slow and gradual stages, never making a sudden step. There is no point where we can say, Here is mind for the first time. For such a development, including, as it does, an organic as well as a spiritual evolution, we must presume, as Weismann has taught us fully to understand, that nervous tissue, capable of indefinite multiplication, carried with it from the first all the possibilities which it has since achieved; and that it is now potent for whatever it will achieve for all time. The modifications which it undergoes in each individual, and which are characteristic of the species, are not lost as evolution proceeds, but only become subimposed. All previous modifications are preserved—those which are vital as still active differentiations, but many of them as vestiges or rudiments. These functions necessary to organic well-being are developed in embryo and are mature long before the intelligent functions. Intelligence does not take note of them as subjective, does not embrace them within its experience in adult life, though we may conjecture that infant consciousness is aware of them. The embryo, or at least the infant, has perhaps sensibilities which become lost as others are acquired. In particular, it would seem likely that visceral sensibilities exist at an early stage and are lost when the special senses become actively engaged, just as we know infants have movements which they subsequently lose. Be that as it may, it is an obvious fact that, as character develops, the mind undergoes a great integration, carries forward only a few things in life and drops all the rest. And the important thing for our present purpose is to remember that, though nine-tenths of experience drop off the *mind* and leave no trace in it, it is not so with the brain-cortex. Facts drop into the brain, not off it. Consciousness in adult life is habitually fragmentary; even vivid experiences are attended to only for a time; people are aware of only one thing at a given moment. The mind, therefore, can only be understood as grasping one small bit after another of a vast range of possible things which are for the most part unconscious. Personality grows as the individual conceives of himself as the same person in a series of ever-increasing relations; it is strong according as some definite purpose dominates all experience; and cerebral differentiations corresponding to such integration must be presumed with special reference to inhibition. Personality then is as much an affair of brains as of the intelligence; and its disturbance in insanity is to be regarded as the exhaustion of those nervous mechanisms which represent purpose and purposive conduct

and the consequent revival of irrelevant activities. These things must be borne in mind as we try to interpret the facts of biology, as well as the facts of experience.

Of the consciousness of the lower animals we can say very little that will be generally acceptable. For the beginning of organic reflexes we must go right down to the most primitive organisms, whose nervous function apparently is entirely alimentary. Their nerves respond only to food; their movements are concerned with the appropriation and with the disposal of it. At first there is no perception of food as distinct from rubbish, and we may question if even fairly advanced organisms perceive their surroundings. At such a level we can learn but little, except to remember that there was a time when animal consciousness, if it may be called so, was directly, particularly, and wholly visceral. And this visceral sensitiveness extended, we may suppose, to the respiratory organs when they appeared, to the cardiac, to the reproductive, and to the others in turn. Of such beings it seems mistaken to speak as if they had a personality, for that word implies a conception of oneself in relation to other things; and these creatures are probably not conscious of themselves, but only of parts of themselves. But we may speak of them as having a character—a visceral character. Right up the line to mammals the fact is in general apparent that the nervous system in certain cases, and presumably consciousness, have reference primarily to what is happening within the organism in all its parts—a particular subject-consciousness; and the first outward reference which the organisation seems to have is towards things which have an almost purely nutritional purpose. This preponderance of internal consciousness is carried up even into the big mammalia, is especially notable in some of the domesticated varieties, and in my belief characterises in considerable degree even some human individuals. It is probably an error in interpretation of a cow's movements, for example, or of a sheep's, or of a pig's, to imply any great perception of the environment. The history as well as the anatomy of the herbivora suggests that the feeling of the animal is still very largely visceral. The character of these animals can best be described as still nutritional, with the addition of more or less acute phases of sexuality. Attention to stimuli arriving by the special senses is but an interruption of a consciousness which is still chiefly determined by internal stimuli. And to such a mode of consciousness, with its characteristic over-attention to the bodily states, insane patients are often reduced. Indeed, we are familiar with it in the hypochondriac. But its most obvious revival is in visceral melancholia and in delusions of persecution which refer to bodily symptoms.

In relation to defence and offence, especially in the carnivora, the rare development of special sense—eyes, ears, and nose always on the *qui vive*—betokens a range and activity of object-consciousness, which of course has developed gradually, but which now for the first time is of the most obvious importance. It would be more true, for example, to speak of the character of the fox, the hyæna, the leopard, as predatory or fugitive, offensive or defensive, rather than as visceral. We may regard their consciousness as the converse of the visceral — a habitual attention to the environment, of which internal stimuli are an interruption. That preponderance of outward relation has of course persisted; and there are individuals in whom it is notable—persons who are uncomfortably aware of the smells, the sights, and the sounds of their neighbourhood; or who, without discomfort, are always noticing things. And of either kind of consciousness we have to note how little the attention really grasps of all the things which the brain carries. Every creature has its mode of consciousness, which largely determines the range of its attention, and all its other nervous processes are subconscious.

It is, however, in the social functions of animals—whether merely as a family, or in a flock, or school, or herd—that we begin to have a suggestion of an evolution suitable for the development of personality. Still capable of much visceral sensation, still wide awake to environment in general, the attention of animals in a family or flock has, for the first time, an opportunity of relating to systematic activities. Now becomes manifest the domestic consciousness, with all that entails of attention to each other, and upon which the whole fabric of social feelings is based. In such conditions also arrives that kind of object-ward attention which is an advance upon the merely predatory or fugitive, which relates to the environment as amenable to work—the construction and care and fortification of the home, the colony, the city. In this is the beginning of the pre-eminently human faculty, one which, not content with the world as we find it, proceeds to a reconstruction of it. The prime character of early man — so that Carlyle distinguishes him as tool-using—is his constructive faculty. In the evolution of these activities which regard the world as a place to be made fit for human habitation is the beginning of all that phase of the mind which we may speak of as purposive. Organisation, providence, ambition, ideality, became possible only as men regarded the world as a place to be taken in hand—different from the point of view of the self-regarding beast, which takes things as it finds them, except when it destroys them. This constructive mode of consciousness is of course still characteristic of our race—so much so that every generation sees a new face upon the world, an evolution of the environment to which the individual must be educated. And, in insanity, when

purposelike activities are no longer possible, the fashioning of demented tools, and futile constructiveness of various kinds, are quite common.

In social life also comes the opportunity for the thinking consciousness—a phase of mind which we cannot recognise in any considerable degree on a lower plane. The first form of it was probably very practical—the thinking out of means to secure very near ends. But only in the security of a society was there possibility also for the speculative form of thought—the kind of intelligence which, curious and filled with wonder, began to ask questions and to seek a reason for things. That kind of mind has probably become rapidly more common with the pacification of the world. It is to be noted as in a cycle which, leaving the more abstruse problems to the philosophic aristocracy, comes back again to the practical problems with which science deals.

But the important fact to note concerning early gregarious life is that thought and feeling and purpose were probably determined by what we may call the subordinate mode of consciousness—one which looked at things from a social and not from a strongly individual point of view. Each man and woman grew up to regard the tribe as the main thing, and the self as of subordinate importance. But primitive man fell away from this mode of consciousness, when he began to regard himself as more than all the rest. When, in a society made safe and comfortable by combined effort, man orientated sufficiently to regard himself as apart from his neighbours, personality as we understand it began. Departing from tradition, men here and there achieved the conception of a personal career; and in that conception, other men and women, as well as everything else in the world, were to be made use of. That notion of a career —from the most humble to the most proud and ambitious—is implied in the habitual human consciousness of to-day. And it is in the struggle between each other that the greatest stress falls upon the organism. When men learned this too great self-regard, insanity as we know it became imminent. For in the sense of individual responsibility and in the sense of personal failure are the greatest subjective occasions of mental disorder.

In polite society, however, no matter how occupied a mind may be with a personal career, good manners and a civilised morality have become essential. These are often lost in insanity, and the consciousness frankly and rudely disregards other people. Says Maudsley: "By insanity of mind is meant such derangement of the leading functions of thought, feeling, and will, together or separately, as disable the person from thinking the thoughts, feeling the feelings, and doing the duties of the social body in, for, and by which he lives. . . . However it has come about, whether by fate or fault, he is now so self-regarding a self as to be incapable of right regard to the not-self; altruism has been swallowed up in a morbid egoism." That denotes what, in more purely psychological terms, Bevan Lewis calls "the rise of subject-consciousness" in melancholia. For it is chiefly in melancholia that reduction occurs from the subordinate to the self-regarding consciousness. In other insanities, notably in moral imbeciles, it is obvious that the subordinate consciousness is still beyond the reach of many. And in perhaps the majority of men a domestic ideal, which still does not have great regard to the good of the race as a whole, is the utmost attained to. Yet is our evolution, if anything, a return, on a higher plane, to that primitive but very moral purpose which determines to think most of people in general and not of oneself in particular.

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But besides all that the human brain has acquired from its ancestral experience, we must take note also of its prospective developments. I have said that all possible evolution of human brain was inherent in nervous tissue from the first, and that the nervous system embodies in some degree not only all the unrecognised memories of previous experience, but also the potencies of all that awaits human character. Whatever men and women are going to be fifty, or a hundred, or a thousand years hence, is inherent in the brains, though not present in the minds, of to-day. For the unique quality of nervous structure, as compared with other kinds of tissue, is its plasticity, that it is apt for differentiations of infinite multiplicity appropriate to any and every condition of life. Men's minds grow and change in relation to an environment which is steadily becoming more complex, and in that evolution an unlimited plasticity of cerebral structure is implied. And while, in average mortals, new character only comes in response to altered conditions, the foremost of the race are those whose personality is precocious, whose minds and whose brains are in advance of their time. Here again we must suppose an automatic maturation of nerve-structure, a preparedness of mechanisms for conditions which have not yet arrived. And that precociousness of brain is not only characteristic of genius, but pertains in some degree also to all who are of robust cerebral vitality, and is pre-eminently the quality of the imaginative mind. As a quaint conceit we may suppose that some people are born into the world which does not recognise their genius, and which calls them insane, persons who perhaps know, or who at least surmise, a great deal too much. A great many quite intelligent mystics believe that we are on the eve of astounding spiritual developments, that the coming race, for example, will develop sensibilities which will transcend the material limitations to which we now

submit. A considerable number of the insane suggest that they possess these occult powers. Apart from these, it is certainly the case that many patients, in contrast with the moral imbeciles, find their way into asylums as to a refuge from an ungentle world not civilised enough for their too self-sacrificing natures.

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These then are the modes of consciousness to which the physician must have regard if he would understand either acute insanity or the much more common and not less important cases which will be described presently under the term, abnormal mental variations—cases of abnormal cerebral development manifest in all forms of eccentricity. They are modes of consciousness which have been arbitrarily emphasised, and we might elaborate upon them indefinitely. I have selected them not only because they seem to designate well-marked biological stages, but also because they become manifest in types of insanity. Melancholia, for example, is a relapse to an almost exclusive self-regard, a loss of outward relations. Mania is the converse—an excess of objective attention, either as exaggerated and incoherent notice of what is in the neighbourhood, or as extravagant outward activity, and generally both. Dementia, if anything worth calling consciousness survives, probably denotes a relapse to the visceral phase, which is also conspicuous in some melancholias. Delusion is an unrelated survival of primitive thinking. And in insanity the less systematic modes of mind also hold the field. Nothing is commoner in our asylums, for example, than the fugitive idea; a bald and unblushing devotion to the domestic ideal of primitive man is common enough; predatory instincts are laid bare in many chronic insanities; sensory hyperæsthesia is much more common than most physicians are aware, and is probably very acute in many neurasthenic melancholics, and in delusional and hysterical patients; daft constructiveness, outvying even the futility of a benighted beaver or ant, constitutes the entertainment of many a happy dement; and less advanced cases spend long hours in inept imaginings. With less assurance we may suppose that some of the over-activities of our patients are modes of thinking and of feeling appropriate perhaps to a stage of evolution still a long way hence. And these modes of attention are still more evident in the abnormal, still short of certifiably insane. Any observant physician will be aware of a large number of acquaintances whose minds are abnormally visceral, or predatory, or fugitive, or constructive, domestic, self-regarding, given over to futile observation, or lost in inept speculation. For the mind of man has not altered very much in kind, but only moves on a different plane, and has to concern itself with new kinds of things.

(2) Personality and Subconsciousness.—In attempting to conceive the cerebral relation which mental processes admittedly have, the physician should be guided by his familiar knowledge of the interdependence between brain and mind on the level of simple functions. We all habitually think of vision or of hearing, for example, of articulation or of penmanship, as dependent upon cerebral activities on the Rolandic level. Yet these are, or may be, mental processes. And what is true of them is true of the higher functions. Integrity of the præ-frontal cortex is essential to proper feeling, to sound thinking, to right conduct. It is equally futile to explain the mind as independent of cerebral processes, and to explain it as wholly determined by them. The relation is one of many of an analogous kind, much more subtle than that of simple cause and effect. But a great deal of unnecessary mystification has been made of it. Not that there is no mystery. But there is certainly no greater difficulty than baffles us when we attempt an account of other ultimate processes. An erudite account of a telephone system, for example, really explains nothing satisfactorily. In the end no one understands the relation of the voice to the wire: and that is the kind of mystery which surrounds the relation of mind to brain, and the only one which is at present quite beyond our horizon. For the cerebral function has to do with forms of energy and nothing more. Brain is simply the vehicle of words and of other things heard or seen or otherwise perceived — energy which travels hither and thither in the cortex, to reappear in conduct. And to appreciate this relation of human energy the physician might do worse than think out this analogy of the relation of life to the telephone. For the telephone and the phonograph do just what the brain does, though by different means. Especially when we learn that the telephone and the phonograph have been combined—adding a function of retention to that of conduction—is there aptness in the simile between the electrical and the cerebral mechanism. Both instruments, for example, depend for their function upon something which we may call tone—electrical in one case, nutritional in the other. If the cortex is not nourished it will not conduct properly; but in health the nutritive processes maintain the lines in good order for the passage along them of the appropriate currents. Further, both instruments depend for their efficiency upon correct and intact connections. Every little while interruptions occur because of wrong contacts, and the energy is diffused in wrong directions. One cannot insist too often that all the positive signs of insanity are of this nature. We have been too slow to appreciate the lessons to be derived from maladies of the sensory and of the motor areas. These we have long ago

learned to consider as manifesting paralysis—loss of function; and diffusion—disorderly function. Excitement, delusion, fear, and other positive symptoms of insanity are but incoördinate excesses following upon a paralysis of those mechanisms which normally command the cortex. There is no difference in the kind of effects which disease has in insanity and those which it has in tabes, in multiple sclerosis, or in a lesion which affects Broca's convolution. The difference is only one of situation. In each case the defect is that normal connections have failed and movement passes by wrong channels.

We must, however, take note of a feature of nervous tissue which is essential to subconsciousness. Next to its conductivity, which is its first and obvious function, the brain is an organ of retention. And retentiveness depends upon the nutrition of differentiated structure, just as every other function does. We are very liable, if we think of our brains at all, to conceive only of that function which is engaging our attention for the moment, or of our habitual activities. But, if memory has any organic significance, it implies that every experience to which we attend—and others as well—leads to a characteristic differentiation; and that the mechanism of it is maintained indefinitely and is functionally active to some extent all the time. To furnish an image of something that we saw or heard an hour ago, a year ago, twenty years ago, the brain must have preserved an activity in a definite and appropriate set of nerve-cells and fibres. One must therefore conceive of the mind as afloat upon a vast sea of movements, of which only a small part comes now and again into relation with it and engages the attention. Here again we may revert to the analogy of the telephone, and remember what an ocean of words is streaming all day long through its countless wires. Sometimes, just before the contact is made for you which isolates the message that you want to hear, a hum as of a million voices vibrates in the instrument until suddenly the proper voice is focussed. In the cortex a similar relation obtains. From infancy onwards, personal experience has initiated a million activities which persist indefinitely in the brain. Moreover, other cerebral activities which are independent of individual experience occur automatically—instinctive processes which are the inheritance of the race. Axioms of life, for example, self-evident propositions which the mind accepts as facts the moment they are presented to it, imply a cortical structure which was active before the occasion for its use arose, just as the act of sucking implies an automatic maturation of a motor mechanism. But both these activities which experience initiates and those which are instinctive lie buried, for the most part, beneath our consciousness. They come within the attention only as occasional facts, when contact is made with them in the course of a train of feelings or of ideas, or when an outward stimulus occasions our notice of them. Subconsciousness is as vast and as real as are the unheard, because disconnected, messages of a telephone system.

We may compare this development and differentiation of cerebral mechanisms to what occurs in the growth and development of bone. Nerve-cells and fibres maturate and become differentiated very much as bone cells do. There are to begin with what we may call centres of cerebration—foci of developmental activity, which are common to the race, and whose diverse proportions determine individual variations. For example, the centres of vision, of hearing, of sexual feeling maturate automatically at different times, and character is modified according as one or other of these (amongst a host of others) is strong. But structure is determined also, as in the skeleton, by functional factors. What the mind attends to, that will the brain become apt for, as surely as stress moulds bone.

The relation of the mind to subconsciousness has been made the subject of much unnecessary mystery. We need achieve no new conception in order to understand it. If we analyse our mental processes we shall discover that attention to an idea or to an emotion is not different in kind from a simple sensation. If a thorn pricks, we are aware of the prick simply as such without thought of the rest of our body. And attention to a mental fact is equally simple. We attend to a fancy which jumps into our mind, or to a memory, long hidden, which something at hand suggests—both of them derived from subconsciousness—just as we attend to a prick. The difference is that we do not locate the object of our attention in the former case. We have not developed a mechanism for the location of mental processes. As I have suggested, consciousness has spread in its development as each system after another became the subject of nervous function. When at last nearly every part was possible of discernment, the mind was in a position to perceive the body as a whole. But there is nothing different in the perception of one's own body and the perception of any other. That kind of self-consciousness then—the bodily self-consciousness—is nothing strange. Nor is it strange that we should be aware of nerve movement which is occurring in our brain any more than of one which is in the big toe. The only difference is that we do not locate the former. The nervous activity which underlies an idea simply does not appear to us as occurring in any particular place. Hence of course the difficulty of realising that mental facts have physical facts to correspond. But the brain is one of many viscera whose nervous activities we do not locate. (No healthy man locates his pancreas.) None the less of course the experience, the

content, which accompanies cerebral activities, is as real as that which we recognise as having to do with a toe or a finger. And, as the mass of these unlocated cerebral facts multiplied, men and women — or at least some of them — have come to regard their spiritual selves as themselves and their bodies as secondary. Character or personality has evolved ever since that time. With the spiritual aspect of such an evolution we have nothing now to do. Our business is to remember that every tradition appropriate to the race has had a nervous structure to correspond with it in the brain of each individual who accepted it; and so also with every personal experience. At school, in the family, at college, in the workshop, traditional discipline is constantly at work, and but for the force of individuality, moulds all men and women to something very near a given pattern. And as men think, they choose as a rule the kind of life which their training has suggested, with just a slight modification due to the personal equation, so strengthening by act of will the force of education. All that process simply implies a very definite and very stereotyped kind of nervous differentiation. It implies that in each individual the great mass of cerebral activities hardly come into contact with personality at all; and, conversely, that those activities which the man selects as appropriate to himself become strongly integrated. A man's brain is commanded by the functions to which he devotes his attention. And such a stability is to be expressed physiologically in terms of inhibition. Inhibition is a nervous process which we all recognise in what we may call its focal form in such mechanisms as control cardiac, respiratory, cystic, and rectal movements. In these an isolated and recognisable group of cells and fibres control the activities within their physiological sphere of influence. But the process is one which pervades the whole central nervous system. Indeed we cannot understand cortical development and integration at all except as a serial evolution of one function after another, each having a sphere of influence in which subordinate mechanisms are under its control. And in the mechanisms of the stable mind this process is one in which every cerebral activity which comes within the province of the intelligence is under the inhibition of the centres which represent personality.

We are now, then, in a position to state less vaguely the relation of personality to subconsciousness — a conception without which, or some substitute for it, any general understanding of sane or insane life is impossible.

Subconsciousness is that whole mass of cerebral activity to which the mind does not attend. At any given moment that is nearly everything that is occurring in the brain. By means of attention the mind handles, so to speak, one of the brain threads at a time, or several in very quick succession, does something with each, and drops it again. Some parts of subconsciousness never come within attention at all; some come once into it and never again; some are very frequently there. Further, the mass and kind of subconsciousness varies with different constitutions. And it varies, and the mind's relation to it varies, with individual experience and habit. The essential point is, however, that at the moment of contact, when the mind is conscious of a cerebral activity (not *locating* it), something happens. As the mind moves along a train of thought, for example, the thread of cerebral activity which it pursues is not left the same as before. Next time it comes into consciousness it is different. The irrelevant and casual things to which we attend we need not now consider, except to say that their subconscious existence explains, for example, a vast deal of the sayings and doings of the delirious and of the insane, who fetch forth from their recesses many strange and hidden and sometimes horrid things with which their friends never knew them to be acquainted. But a great deal of life is consecutive and coherent, and becomes woven into the personality. To become concrete, take the commonest and the average personality whose central feeling and idea and purpose concern themselves with livelihood. At the foundation of that there is the domestic consciousness —wife and children, the hearth, bread and butter. For that there is certainly a cerebral mechanism—a centre diffuse in form, but definite in structure. We have not located it yet, but surmise that it is in the præ-frontal region —the area for the images of acts; for personality is determined by its expression of itself—what a man conceives of himself as doing and saying. All consecutive experience then comes into relation with the man's notion of livelihood. From religion to silly pleasures the criterion of their effect upon his position and comfort and prospects is the constant one by which the man gauges things. And that means a great integration as well as a great spread of mechanisms. The process is one which occurs to some degree in every mind, for even the most versatile have one or a very few notions to which they most attend. All through life, then, one activity after another is coming into relation with these highest and most central mechanisms. Each point of contact modifies both. On the one hand, the primary mechanism is modified, extending its fibres so as to meet and, if all goes well, bring that other within its sphere of influence. On the other hand, the secondary mechanism is modified conversely, just to the extent to which it has been subordinated to the primary centre. A sane and successful life is one in which every conscious experience enlarges the range and adds to the power of the organs of personality. The occasions of insanity, over and above the organic causes which interfere with nutrition, are those

experiences which are in conflict with the main purpose of a man's life. To revert, for example, to the average personality mostly concerned with the hearth, the commonest occasions of insanity are domestic affliction, or domestic dispeace, or financial troubles, or social disgrace. It is friction which breaks a man's spirit—when the circumstances of his life thwart his purposes, or when impulses bred of vicious organisation or wayward habits pull against them. His brain is an arena of conflict between activities which he has made his own and others which are being forced upon him. Then, if personality is not strong enough and purpose fails, the attention becomes engaged with the irrelevant and perhaps the insane things of a subconsciousness that is without rational direction or supreme government.

II. Index of Mental Functions and Symptoms of their Disorder

It is a very doubtful question if psychology can ever become very scientific, and if the knowledge of the physiology of mental processes can ever be exact. The chief difficulty in the way is that analysis is futile. The solidarity of the mind is too great, the brain is too compact, to admit of dissection. Hence all attempts to describe the faculties of the human mind are misleading, and even descriptions of particular processes are rarely convincing. But some attempt must be made to indicate the most important aspects of mind, or, as physicians should regard them, the mental functions of the brain. Even if our description of them fails of reality, it is still essential to note certain kinds of processes which one should habitually observe.

Instincts.—An instinct is an automatic activity, the common inheritance of the race, made manifest in appropriate movements, and, in its higher forms, realised in appropriate feelings. Instincts for vital movements are familiar. One need not enumerate them. But comparable to them, at least in their physiological aspect, are other less elementary instincts which subserve a social function. These also betoken an innate development which, in every normal brain, is more or less mature before the occasion for their manifestation arises, although, as always, practice makes them more perfect. An enumeration of these must needs be arbitrary and incomplete; but we may note those of which we most commonly have to observe the failure. The instinct to prolong one's own life and to guard against danger comes first. An arrest of its development constitutes the predisposition to suicide, which is often hereditary; and homicidal tendencies mark those who fail of the instinct to respect the health and life of others. Pain and discomfort are generally the occasion for their active manifestation. The normal man is so constituted that pain and discomfort, or the knowledge of them, prompt him to alleviate them. Mutilation of oneself, starvation, and the many degrees of disregard of pain and of discomfort, all very common in insanity, betoken an impairment of the life-preserving instincts. The mating instinct is another, whose arrest, or excess, or perversion so commonly appear as signs of cerebral disorder. Next in importance, perhaps, comes a group of instincts which we may call the domestic—instincts which have developed in relation to family life. Whatever happens, the instinct still persists which attaches us to our own folk; and few symptoms of mental disorder are so tragic as that perversion of the strongest of the domestic instincts, the maternal, which culminates in infanticide. Going beyond the hearth, the step is easy to that instinct which prompts men to get on in the world, and to that other which enjoins upon us to seek the company of our fellows, and to earn their favour. Departures from these, or excesses of them, are commonly to be noted in disordered minds. And lastly among the social instincts we must take note of a patriotic instinct, the highest perhaps of all, for, although we may conceive a cosmopolitan ideal, we can scarcely be said to possess a cosmopolitan instinct. The care for our country and for our race is certainly inbred; and, though we do not often have occasion to note a failure of it as a symptom of actual insanity, it is interesting to observe in how many unexpected quarters recent events have made evident the defect of it. There still remains the religious instinct, the instinct to reverence and to worship, which, whatever we may think of religions, is the subjective basis of them. That instinct, as every one knows, often finds abnormal expression in disease or falls into abeyance.

It were easy to multiply the instincts and to refine upon them, but those enumerated are sufficient to suggest the others. They constitute the foundations of well-organised brains. Any lack of them, any perversion of them, any extravagance of them, betokens disorder; and in gauging his patients' mental health the physician must take careful note of them. And let me repeat for the last time that we must conceive of a development of mechanisms to correspond to all of them,—a maturation of cells and fibres which represent them,—and a subconscious, occasionally conscious, activity in these instinctive centres. These are the primary points of cerebration, so to speak, whose automatic activity determines personality.

Interests and Tastes can be more summarily dismissed, for the reason that they are more varied and less universal. But it will be obvious to any one who has watched the development of children, or who has thought of it, that innate cerebral organisation determines to a large extent the preferences of the individual. There can be no doubt, for example, of a specialised æsthetic

taste in some cases, of an innate greed for rich colour or fine form or good sound. A useful observation has distinguished between the ear-minded and the eye-minded—those whose brains have the avenues of hearing or of vision specially active; and, more commonly, we have to observe practical tastes—an interest in making music, or in producing colours and forms, and various other outward activities, of which the love of sports is perhaps the most usual. In a well-balanced mind æsthetic interests are well blended, but the normal development is always towards practical pursuits. Short of the gross defects of vision and of hearing, and the gross motor failures with which we are all familiar, slighter degrees of defective interests are often to be noted as characterising bad brains; and, in insanity, inquiry always elicits an account of a change in the patient's pursuits.

Humour, Temperament, and Disposition.—These constitute still another kind of innate cerebral characteristics. The day of temperaments is past. But we have still to note that many people fail of that good humour with things in general which is the good fortune of a healthy brain, and that, in active disease, an extravagant optimism is common. So far as is known, good humour is determined by a right proportion of expenditure and revenue of energy. It is when the brain has enough vitality and to spare for all the calls made upon it, and—what is apt to be forgotten—when it is allowed to expend its surplus, that good humour comes. And temperament is determined by the prevailing humour. The term disposition merely indicates the expression of temperament or of humour. It signifies general feeling made manifest in conduct — *bonhomie*, kindliness, tolerance, or surliness, sourness, moroseness, hopelessness, as expressed in converse with our fellows. In all disordered states of brain the disposition is changed, though often pleasantly as in the early stages of mania.

Sense-Perception.—The functions to which allusion has been made are functions in which the innate constitution of the brain is obviously of the first importance—functions which depend upon the original vitality of its parts. And it is still true of those which we are about to consider, that development, up to a certain point, precedes experience. But experience now plays a more obvious part. If the eye is not filled with seeing or the ear with hearing, it is still true that sense-perception is a function or group of functions which depends very largely upon experience for its development. And these functions, which we are too apt to regard in their primary aspect—as merely subserving sense impressions—furnish very largely the data of intelligence. We think and feel and act after we have seen, heard, tasted, smelled, and handled. We must, therefore, conceive of each sensory tract not merely as developed in relation to the objects to which it obviously refers but as woven into all the mechanisms of the intelligence. To conceive the nervous basis of mind, we must think of an extension into the mental areas of these mechanisms which we commonly associate with acts of simple observation. The visual system, the auditory system, and all the other sensory systems, are essential elements in the framework of intelligence. And in that conception we must include the visceral tracts whose activities in health do not obtrude themselves upon the attention. The ovary, the heart, the intestines, are intimately related sensorily with the mechanisms of feeling, and constitute a large area of subconsciousness. Disorders in all these realms are familiar—paræsthesias, illusions, hallucinations. Sometimes the disordered activity is high up among the mental mechanisms, sometimes in what is usually termed the sensory area, and sometimes peripheral. It is not always easy to discover the extent of defective sensation in mental cases, but there are few in which, if the physician seeks for it, he will not find some disturbance of function. (It may be noted here in passing that there are equally intimate and elaborate relations between the mental areas and the motor tracts. Activities in these connections, however, are chiefly outward, and disorder is more likely to spread from mind towards muscles than contrariwise.)

Memory.—One of the prime characteristics of brain as an organ of intelligence is the fact that it retains some kind of image of the impressions which it receives. This function of retention, though most obvious in memory, is implied in all mental processes; or, in other words, memory comes into every act of the mind. As has been said, attentive observation does something to strengthen, that is, to make more facile, the images which the brain stores; for attention generally implies that we are associating the impression of the moment with some train of thought or feeling which is more or less habitual. But we do not know that attention does more than that. Probably all impressions which reach the brain—and they are innumerable—have their characteristic effect; but, if not associated with personality, go to swell the mass of subconsciousness. That this is so, is supported by the facts of delirium, in which the brain reproduces innumerable things heard and seen which had lain quite without the voluntary recollection of the patient. It is indeed doubtful if the brain evolves much material for conceptions, though the process is not incomprehensible. More likely the new combinations which occur, for example, in dreams and in active imagination—new scenery, new incident, new personality—are chiefly compounded of images fished up from subconsciousness and strung together. But the products of retention are more orderly associated in rational memory. And here we must observe a fact which comes second only to retention in its im-

portance to character—that each stimulation of mechanism (short of fatigue) increases its facility. Practice makes perfect; familiarity deepens impressions; repetition strengthens memory. As I have said, any retained image which can be recalled probably implies a mechanism in which there is constant, though subconscious, activity. But, perhaps for want of renewed impulse, these currents, it may be, lessen and cease; while those habitually augmented by restimulation are perpetuated. And the mechanisms can be no other than the same mechanisms primarily concerned in perception, or perhaps an extension of them. To learn a fact and to recall it imply very comparable processes.

Different degrees of association and of repetition characterise the distinction, useful to the physician, between old facts, recent facts, and permanent facts. In investigating a patient's memory we try to discover how far he can recall the facts of yesterday, or the facts of ten years ago, or again a fact which has been all along the same—as, for example, the Queen's name, the date of Waterloo, or where the baby Moses was found. In all mental disorders there is some impairment of this function, and in a few, especially in alcoholic and in delirious insanities, the defect is quite characteristic. It is chiefly in disease that we discover also the importance of the observation of three phases of memory—retention, recollection, and recognition. Defects of the first of these alone are practically unrecognisable. But it is very easy to detect defects of recollection, as, for example, in a patient who observes and clearly apprehends a fact, yet five minutes later cannot recall it. And of recognition—the function whereby we recognise the recollection as the thing we have been seeking for—disorders are common, and most obvious in cases characterised by mistaken sense of identities. A very interesting phenomenon, most familiar in the senile, is an extravagance of recollection which we may call reminiscence. The patient lives and moves in scenes of fifty years ago, the attention dwelling upon images which may have lain subconscious for half a lifetime; and, in minor degree, the process is common in many disordered brains. Lastly, we have to note a very difficult function—prospective memory, the ability to remember forwards. This function is one closely related to purpose and intention, and is very commonly, and often early, disturbed in mental disorders. The inability of the insane to carry forward their plans is often more notable than their disability in conceiving them.

Thought, Intelligence, Common Sense, Beliefs.—The fact that so few people can really be said to think, taken with the average amount of level-headedness and sagacity, and the average amount of proper conduct, speaks volumes for the subconscious activities of brain. In the rational sphere common sense is to knowledge what the domestic instincts are to the affections. Most of the sagacity of the average person is innate or intuitive knowledge, and not the fruit of logic. This implies then an abundant activity in nerve mechanisms which automatically determine opinions and beliefs. Thought indeed, in its elaborate modes, does not often call for investigation in mental disorder. It is a delicate process which, in those who have practised it, disappears very early in disease. In its place we have to note, on the one hand, premature inferences, and, on the other, extravagant imaginings, both alike due to an abeyance of corrective judgment. The foolish and the deluded seem to accept blindly almost any opinion that their brain serves up to them; and that this disorder is a product of subconsciousness is testified to by the fact that these people invariably prefer their own explanation of things rather than any, however plausible, suggested by an outsider. The physician, if he tries, will find it hard, and hardest in the most deluded, to substitute one insane belief for another in the mind of his patient; those mentally disordered are notoriously not amenable to suggestion. Delusions then are generally intuitive products, although circumstantial facts may later be adduced to support them, and are similar in kind to the imperative ideas which we call obsessions. In obsession, the brain presents persistently to the mind an idea, often quite irrelevant to the rest of the patient's life, but eventually touching his personality. These phenomena partly indicate a disordered activity from which some imaginative opinion rises into consciousness and dominates it; but they betoken diminished rather than excessive thinking. It is the function of corrective judgment, the ability to weigh the facts carefully before coming to a conclusion, which is at fault. Gullibility is a very common feature in persons who are of the insane diathesis; and a sense of probability is conspicuously lacking in the insane. These defects obviously betoken a want, or an impairment of common sense. Short of delusion, the practitioner will often observe minor failures of this kind—facility, opinionativeness, extravagant expectation—due to an exhaustion of judgment, and likely to be followed, if not corrected, by positive delusion. Quite as often also—and this is important—we observe in the unhealthy mind persistent confusion (see later under Confusional Insanity). In one sense confusion is normal to all thoughtful minds; it characterises our state in regard to every question concerning which our minds are not made up, and the more thoughtful we are the less sure we are. But confusion in the insane very often refers to things which, in the sane mind, admit of no question. It is essential to mental stability that we should take a great deal for granted, that we should assume that what everybody believes is true, that we should continue to hold beliefs which we have proved.

Any of us would become painfully confused did we, as these people do, entertain serious doubt as to whether this is really Wednesday, whether it is wise to put on one's boots, whether food is healthy or poisonous, whether we can or cannot do without breathing. Such are the failures of common sense familiar in mental disorder, and this perplexity, in a minor degree, characterises many slight affections. But some observers make the mistake of assuming that patients are confused because they talk confusion. Deluded persons are not really confounded; on the contrary, foolish assurance is much more obvious in the insane than confusion is. Doubt is characteristic of less grave disorder.

Feeling, Emotions, Affections.—The term feeling is best confined to the simple states of pleasure and of pain. It attaches normally, as we know, to certain kinds of experience, to certain ideas, and sentiments, and emotions. As has been said, the absence of distress or discomfort betokens a nervous activity for which the brain has sufficient energy. So soon as fatigue occurs, whether from over-exercise of the brain, or because it is ill-nourished, pain arrives, and the patient is depressed. Then even the experiences which can commonly be counted on to give pleasure fail of that effect. Bad spirits, a defective capacity for enjoyment, are amongst the commonest signs of mental disorder, and call for treatment which will promote regeneration. In some cases fatigue induces the patient to dwell upon his own misery, and melancholia supervenes; or, if self-control wholly fails, he takes to extravagant conduct and becomes exalted and maniacal. In many cases, notably in general paralysis, there is no period of depression of greater amount than is common in sane persons. The joy which supervenes in mania and in general paralysis is to be attributed to the abeyance of fatigued mechanisms and to the activity of the less wonted centres—those which subserve imagination, and the sensory and the motor areas.

Feeling attaches to all emotion and in characteristic tone. Speaking roughly, emotions which add to the possibilities of life are pleasurable, and those which take from them are painful. Hope and fear are types. Physiologically considered, hope opens out avenues of activity; fear closes them. And these states are instinctive, and, in some degree, universal. We cannot fail to note, however, great differences in individuals—that some are instinctively prone to hopeful and others to fearful emotion. And we observe also changes in individuals which betoken impending or actual disorder. Extravagance in either direction is bad, but the unhealthy are, as a rule, fearful.

Affections are cultivated instincts—instincts for people and for things which have become elaborated by contact with them. And here again we must note differences in individuals as well as changes in the individual. Here also the observation is roughly sound that pleasure betokens a healthy mind. In the more sane, likes exceed dislikes, hatreds are few and friendliness common, sympathy should outweigh antipathy, tolerance exceed criticism. In the unhealthy, the balance is the other way, and in mental disorder the affections are gravely assailed. Suspiciousness, antipathy, loathing, anger, supersede even what were strong affections. And, on the other hand, persons not normally affectionate may, when intoxicated with their insanity, fall in love with all and sundry, see nothing amiss, dote upon objects almost vile.

Habit, Impulse.—On the outward side of mind, in the realm of conduct which, as has been said, largely determines personality, habit must be our first consideration. Its persistence is determined by that fact about nerves which we noted under the subject of memory, that stimulation of mechanisms increases, up to a certain point, their facility. What a man's habits will be is obviously determined to a large extent by his instincts and tastes, but not less by his experience and especially by his education. In a sane and well-ordered life habits are part of a symmetrical whole, fit with general conceptions, and do not interrupt or impede the even tenor of a man's way. Vicious habits, on the other hand, are rebellious and out of keeping, fail of the man's general approval, and so engender pain. Even in the well-behaved, disorderly or unrelated habits may be a source of much worry; and there is no commoner source of mental mischief than the constant knowledge of bits of life that are in discord with, perhaps directly subversive of, the whole. It is a truism to remark how easily habits are formed, and here we have to note the converse of what has been said about instincts. While it is true that subconsciousness makes great contributions to mind, it is equally true that a great deal of our personality sinks into subconsciousness. Almost as soon as we have thoroughly learned anything we cease to be vividly conscious of it, though we can perhaps at will make contact with it again. And this process of habit implies that vast numbers of mechanisms, whose organisation came into existence under our direction, fall into a place in subconsciousness in relation to all the others. In sanity the vitality of those subconscious mechanisms which we have chosen exceeds that of others. In disorder the hierarchy is disturbed by the excesses of centres not closely associated with conscious purpose. A man's brain, then, is strengthened by all habits which are consciously related to his general purpose and approved of. We all know, and there is no need to elaborate upon it, what kind of habits make for mental health. We take it as roughly true that average habits are normal, and are justified in scrutinising the irregular. Far more useful, however, than a comparison of

one person's habits with those of another is the observation of change in the individual. And it is a curious fact that mental disorder is generally associated with a change from the correct towards the disreputable. It is not often that one is alarmed by an access of propriety in a patient. But any change, especially in adult life, is noteworthy. In the insane it is remarkable how great is the departure from the wonted, and that often without any apparent conscious impulse or intention.

Regarding insanity, as we must, as a subversion of self-direction and the ascendancy of disordered nerves, impulse becomes very important. But the facts regarding impulse are so obvious that we need not dwell upon them. It is enough to revert to the possibilities that lie under the surface of the human brain. In any of the mechanisms to which we have referred—mechanisms which in sane life are subordinated to a general purpose—an excess of activity, occasioned by illness and the failure of the normal, may spring an impulse upon the unwary mind. And such impulses are as strong as those which are occasioned by normal conditions and which earn the mind's approval. And so, in various insanities, but especially in alcoholism, and in the insanities of the degenerate, an impulse, however preposterous or dangerous, may possess the consciousness just as an obsession may swamp all thought; and, unless prevented, the impulse will be realised in act. Like obsessions, these impulses are to be discerned as they are more or less at variance with the patient's habitual personality. Homicidal impulse, for example, may as a rule be taken to be due to an activity in mechanisms with which the patient has not been much in contact. Less insane impulses are of course commoner, and, equally of course, less observed—impulses to do just not quite what would have been done in health.

Moral Sense and Sense of Fitness.—By instinct, by education, and sometimes by conviction, our minds learn to know and to approve the right. The difficulties that beset any inquiry into the nature of that knowledge are great. But, as physicians, we have to regard moral life as of the same nature as other activities of the mind. Whatever be the code to which we have been born, and in which we have been brought up, our apprehension of it implies the development of an organ to represent it. These mechanisms are no more, but rather less, detached than others; they are, like others, related to all conscious life, capable of contact with the great mass of subconscious processes, and subject to disorder and degeneration. A less dignified function, but strictly analogous, is that which approves what is fit and proper—a function which relates to manners rather than to morals. That our appreciation of the good and of the proper is largely instinctive there can be no doubt. The brain is prepared to react to moral as to other stimuli. Many patients manifest an instinctive disability in this regard, are not easily made to understand what is right, and, when they do understand it, are not convinced. And, just as we have perversions of other instincts, so also of this; and men and women are to be found who are carried away by a zeal for this or that other virtue which the level saint regards as one amongst others. So too, in mental disorder, the moral sense often fails and shamelessness supervenes; or an extravagant conscience condemns fiercely even the most trifling offences. Similarly, the sense of what is fit is so commonly lost in mental illness, that we hardly recognise it as a fact that the insane and the unhealthy-minded are unmannerly; and, on the other hand, we have sometimes to note an excessive punctiliousness as a symptom occasioned by cerebral change.

Purpose, Intention, Ambition, Ideals.—We have already considered purpose in considering personality, and need only remark here that the function of prospecting life and of choosing a line is to be discriminated from that which gives effect to what has been chosen. In mental disorders they may be separately affected. A patient may conceive of life in disorderly fashion, may think the wrong things desirable, and may choose them, yet may carry out his wrong purposes with healthy vigour. Or ideals may remain intact, yet conduct wholly fail to realise them—a disability which is too obvious and too familiar to require illustration.

Conduct.—Downwards and outwards towards the muscles all mental activity eventually trends, at least in the healthy mind. Thoughts are to be spoken, affections to be demonstrated, projects to be realised. And this process of expression of one's personality would appear to be the last and highest function of life, the final task imposed upon nervous organisation. That failures occur in this function is very obvious; in fact nothing else in insanity comes under our notice. It is as the patient looks, and speaks, and acts, that we judge of his sanity or of his disorder. As a rule we infer that a foolish remark implies a foolish idea in the consciousness of the patient, but such an inference is not always just. Just as, in thoughtlessness, we say and do what has no significance to us, so, in mental disorders, much of conduct is without consciousness. In all excited states things are said and done which the patient probably does not choose to say and do. The mechanisms of speech and of deeds have the bit in their teeth and are entirely undirected from above. Or, what is equally common, there is simple abeyance of expression. In mild disorders nothing is commoner than for the patient to complain that, while he can quietly and properly prospect what he should and will do, he finds to his dis-

may that he cannot do it. This is in particular the symptom of stupor, which is essentially a depression in the functions of expression. Sometimes stuporose patients remember what they have come through, and will tell you that they were labouring to say and do things, and failed. The contacts between the mechanisms of purpose and of conduct had not been made, or, if they were made, the tracts of conduct would not transmit currents. Lastly, we have to note that most interesting phenomenon, resistiveness, in which the nerves, not content only with not doing what is wanted, insist upon doing the opposite. From the experience of the golfer, or other artist, who finds his performance so out with his conceit of it, to that degree of resistiveness in which the patient becomes rigid at the merest suggestion of movement, all degrees of resistiveness are to be observed in mental life.

The Self in Insanity.—In sane life men control themselves, which is to say that they keep those parts of their brain in subjection which they prefer should be so. Insanity is the reverse, it is the subjection of the mind to a disordered brain. That is why it is fitting that, for purposes of insanity, one should regard mind in its organic aspect, so as to understand and be prepared for cerebral effects. It is true beyond any doubt that cerebral occasions mental activity; but it is no less true that mind influences nerves. It is quite obvious that nearly the whole brain can be brought into subjection to purpose and intention. We are then face to face with the problem of what becomes of the self when mental disorder has usurped it. And I have no desire to contribute to metaphysical futilities on this question. But if this relation of mind to brain is a fact, it has great importance in mental disorders. And I would ask the physician to revert again to the analogy of those incoördinations—such as aphasia and ataxia—which we know to be due to motor lesions. In such a case it may be the patient is aware that when he intends one thing his nerves and muscles bring about another. So in insanity there are no doubt cases in which the patient realises that these words and acts are not his. In most instances, perhaps fortunately, insanity is as sleep, or as a dream, and the patient remembers not at all, or but vaguely, what has happened. But in all cases of convalescence the mind clearly resumes authority, and in many cases, it may be, it has been conscious of its overthrow, and not without hope of restoration. To build upon that, or to try to explain it, is to embark upon sheer mysticism. Yet the physician who prefers to be a mystic will not fail of reward if, while he does all he can to restore the disordered brain, he thinks kindly of the sad soul who is tethered outside of it, and if, when occasion offers, he tries to reach it and to reinstate it.

III. The Insane Diathesis

I have remarked, and repeated, that the brain has in it the potency of all that human character is going to be, and that it is unique in its plasticity. It is the tissue which, above all others, must be prepared to take on new developments. Each generation must be innately prepared for new ideas and new habits. And such a condition implies a possibility of great fluctuation or variability in the individual. Physiologists sometimes forget that, and omit from their account of brains the facts of the personal equation. For an organ which is still plastic, which is still evolving, is sure to manifest great departures from the type. And, though we cannot discern in every case individual peculiarities of cortical structure, we certainly can observe mental differences. To take an exaggerated instance, nationality makes all the difference. For that reason, I think, we attach far too much importance to descriptions of mental disorders written by foreigners. Not that their observations are unsound, for in many respects they have taught us scientific methods in insanity. But the mind of a Parisian is not the mind of an Aberdonian; a Tyrolese peasant does not think and feel like a Yorkshireman, nor a Dutch cheese-grower like a cockney cabby. And both national and local and personal characters greatly modify types of mental disease. Hysteria in this country is not the gaudy type which it is in France; hystero-epilepsy is a comparatively insignificant thing with us; the mental facts of general paralysis and of alcoholism vary so much as to be hardly recognisable among different peoples; and (perhaps the best case in point) progressive systematised insanity of persecution, as described by Magnan, can hardly be said to occur in Scotland. Even in any one community the forms of mental disease are as important in their discrepancies as in their constant features. No two minds are alike either in health or in disease, and a great many minds are remarkably divergent from the average. And, as we know is generally the case with extreme variations, these odd minds are liable to be unhealthy. A failure to achieve the full *repertoire* of functions which are the possession of the average mind is an obvious weakness, a want. But other minds, not to be termed imbecile, are also weak and unstable. These abnormal variations are brains in which one or a few functions are developed in excess, which means that the mind will not be well balanced. Such excess of function in one direction is apt to be associated with a lack in some other, and the mind fails to achieve that even activity under varying condition which is the sign of cortical stability. These odd minds are in my opinion much more deserving of study than cases of actual insanity. They repay study because, when short of insanity,

they are more easily investigated; because they are of great importance in private life in that they are subject to endless petty disturbances, and are a great trouble to themselves and to their neighbours; and because they are predisposed to acute insanity, and ought to be recognised, understood, and guided.

Abnormal minds we shall therefore consider before proceeding to commonly recognised insanities. And the physician will find it useful to remember a somewhat arbitrary division of them into (1) Arrests of development, and (2) Disproportionate developments—abnormal mental variations or, briefly, odd minds. In most cases there are both elements. But in investigating the mental functions serially, the physician should note concerning each whether it is in excess or deficient.

Arrests of Development.—Arrest of mental development may occur in almost any degree and in regard to almost every cerebral function. We shall consider it as—

I. Partial.
II. General.
III. Progressive.

Partial arrests of development, as was said generally, occur in odd minds in association with excesses of development. One finds all forms of it, but we shall confine our attention to those which give trouble. Of arrested instinct, for example, that form is most important which is a lack of life-preserving activities. Obvious cases are those who simply do not care to live and do not fear to die. But a suicidal point of view is often safeguarded by a physical repulsion on the approach of bodily hurt. More important is a lack of instinct generally overlooked, in which the patient is indifferent to hunger and to fatigue. Many of our insane patients have been men and women who seemed never to know that sleep or rest was necessary, or that irregular fasting was dangerous. And many neurotic people show an analogous disregard of the safety of others. Normal children learn quickly what is unsafe; but many men and women never acquire a proper consideration for the safety of their neighbours, and do not react to a sense of danger to them. Then a great deal of vice and of misery is occasioned by persons who have not a well-developed sexual instinct. Many cases of unhappy marriage and many of sexual perversion are to be attributed to a lack of pleasure in normal intercourse. Like defects of organisation, though not with so obvious a bearing upon insanity, are maternal indifference, paternal disinterest in the home, irreverence or an absence of the instinct to worship, and the failure to eagerly defend one's country without respect to the righteousness of its cause, all of which wants are common in disorderly minds.

As we ascend the scale of mental functions, defects are of less importance from a biological point of view. We expect variations in the advancing points. But all the same some of them are of much practical importance. On the lower levels one of the most important defects is a lack of a capacity for enjoyment. Many good people take pride in their neglect of pleasures, but their failure to achieve a pleased interest in things sometime betokens a constitutional defect. No man who does not enjoy the really good things of life can be described as sane in the full sense of the word. Another primitive defect to which students of insanity should pay much importance is ineptness for muscular activities. Some of the most skilful in technique are neurotic persons; but very commonly those who are of defective brain cannot use their muscles aptly.

On a higher plane are to be noted such wants as we all are familiar with in the sphere of intelligence. Defects of memory are amongst the commonest; so also an arrested development of the understanding. Stupidity—an incapacity for thinking, and a disability in apprehending truth even when made plain—is so common as almost to be described as normal. Special importance attaches to such minds as combine some activity of thought with a lack of corrective judgment. A man who does not think is probably safer than a man who thinks well along certain lines, but who fails to see other sides of the question in hand. Rash judgments are very characteristic of defective minds. So also are truthlessness and a lack of common sense—a defective notion of probability and of feasibility. Odd minds are generally impracticable. Above all these, however, the student should carefully observe those who fail of social quality. The most obvious cases are those in which there is simple defect of sociability. More likely to end in disaster are those who, with a full development of sociability, are devoid of tact. Tact is one of the most interesting and subtle mental functions—a sensitiveness to the feelings of one's neighbours. And perhaps no one sign of the defective mind is so characteristic as tactlessness. Human evolution is towards a fuller and fuller understanding between man and man; and he who fails to realise that he is "rubbing his acquaintance the wrong way," the man who habitually says and does the wrong things, is singularly prone to mental trouble. Many persons commonly set down as bores or as egoists are of the insane diathesis. And lastly, we have to note those who with perhaps no want of manners are morally defective. Failure to develop self-control may almost be said to be a constant feature in odd minds.

General Arrest of Development.—Systematic arrest would, perhaps, better describe the conditions now to be considered. Partial arrest occurs apparently fortuitously in one or another mental function in persons who still pass in

society for sane. In others, however, the mind as a whole has suffered arrest. Or at least there is a serial defect, most obvious in the last and highest functions, though with occasional developments of a disproportionate kind which seem excrescences in a weak mind. Two forms are to be noted: imbecility and degeneracy. Of these, imbecility is considered in a special article (*vide* "Mental Deficiency").

Degeneracy.—On this subject, which has become fashionable of late, a great deal has been said and written which will not stand severe criticism, and the student must take what will be said as a rather tentative account of degeneracy.

As I take it, the term describes a biological conception. A degenerate is one whose mind betokens a failure of his race. So far as he is concerned the race is coming to an end—not actually perhaps, but virtually. A degenerate may procreate children who will prolong his seed indefinitely, but if so, it will be by a lucky marriage and in spite of his weakness. And such an individual is to be distinguished from the merely neurotic, many of whom are of a quite potent cerebral vitality. But if the practitioner sets out to diagnose degeneracy in its mild forms he will certainly fall into error. For the diagnosis between an arrest of mental function which is but a temporary interruption of a family's evolution and that which betokens the beginning of its end, is quite beyond us. And so in practice, at all events, we restrict our use of the term to obvious and gross types, such as are appreciable by an enlightened police. And that is fortunate, for gross degenerates are always immoral and probably always criminal. Dr. Urquhart has indicated the type. But a warning must be given because the subject has suffered a good deal from the amateur exposition of extremists who go much further than he has indicated. In particular, the stigmata, the defects of conformation in degeneracy, have achieved an importance which is altogether fictitious. From scalp to sole no spot or blemish escapes the category of the erudite observer. Degenerates have many of these blemishes; hence, it is argued, blemishes indicate degeneracy—a fallacy which a slight attention to biology, some elementary logic, or even a sense of humour, should have prevented. For we all have good friends who are very ugly; supernumerary digits do not betoken stupidity; early baldness or loss of teeth is not the monopoly of the vicious. The fact is, many of the stigmata of degeneracy are in more or less unessential organs which are undergoing the common fate of becoming rudimentary. As intelligence evolves it is quite likely that the race will depart further and further from a type of physical beauty which evolved centuries ago under the influence of direct sexual selection; and popular ideas of beauty are nearly as fickle as fashions of dress. Once upon a time, no doubt, the disappearance of fur from the human chest and shoulders was regarded as a grave event, and posterity will probably suffer hairless scalps and artificial teeth with equanimity. Surely these externals are of but slight importance, except for police purposes, compared with the internal processes which are essential to nutrition and recuperation. Sterility, susceptibility to infectious diseases, the tendency to other toxications described by Dr. Urquhart, cardiac and other visceral disease, anæmia, vascular degeneration, are much more significant of racial decay than are the anatomical features amenable to the foot-rule. The central fact is, of course, that in degenerates these defects of physique, both external and internal, occur in abnormal degree.

Of mental facts, immorality is the primary and essential feature of degeneracy. Morals are, at least theoretically, the budding point of evolution, and no one may be regarded as degenerate who feels and knows and does the right. And to degenerates lack of morality usually means criminality, for most degenerates are too odd to be law-abiding, and too stupid to escape. In the ranks of society higher than the lowest, however, the temptation to commit police offences is not so great, or, as in the case of sexual perverts, detection may be very difficult. But failures other than in morals are always to be noted in the degenerate. He may be clever, but not of sound judgment; very knowing, but not wise; fond, but not faithful; sentimental, but not just; convivial, but not happy; active, but without endurance. And the practitioner must be prepared to recognise these potential criminals, whether noble or lowly born. His common knowledge of the human features is all the anthropology that he requires for practical purposes; he will pay great attention to facial expression; he will note carefully any defect of nutritional processes; and, above all, he will try to discover, unostentatiously, the feelings and ideas and impulses of degeneracy. And, having assuredly diagnosed these creatures, he will treat them with a view to the safety of society, and with just as much leniency as is feasible. For these unfortunates no liberty, no opportunity of evil, is safe. They must be controlled and disciplined, segregated or banished, or otherwise treated as rigorously as circumstances justify.

Progressive Arrest of Development (Primary Dementia).—The cases now referred to, combine arrest of development with active degeneration. They are cases in which the mind develops up to a certain point and seems to the patient's friends to be going on well. Then it stops, and forthwith deteriorates more or less rapidly. Such cases of apparently idiopathic dementia occur at all stages of life, and they comprise a most interesting group not easily understood. To explain them we must

revert to the idea of a premature senility of nerve-cells which inaugurates something in the nature of a descending degeneration. For example, take the case of a boy at school, a good son and a promising scholar, who, on entering the fifth form, failed to make progress, and, at the end of the term, returned home with his first unsatisfactory report. A year later that boy was obviously demented, had apparently forgotten all he had learned, and was fit only for the work of a common labourer. Some such cases go on to motor paresis. I have under my care now a man who broke down towards the end of his college days after an unusually promising record. There was no sudden change, but only a slowly progressing stupidity, and instead of taking the high place in his profession which was expected of him, he became entirely childish. Moreover, he suffers from a partial hemiplegia, with diffuse motor impairments in all his limbs. His attitude and gait, and especially his speech, are those of an idiot. Visitors who are specialists take him for an imbecile, and strangers cannot understand what he says. Yet there was no sudden lesion, and in my six years' acquaintance with him, his atrophies, as we call them, have not materially increased. Most careful inquiry failed to elicit any suggestion of an active cause of insanity.

Some of these cases are occasioned by the reproductive crises. Precocious senility may occur after child-bearing, or with the too heavy cares of a large household. Similarly, men who become rather suddenly engrossed with large and increasing affairs may not only not rise to the occasion, but may become demented under their burden. And so we can trace the disease right on into recognised senility. Healthy brains are still plastic in old age. Some men and women never know dotage, but others begin to be senile at the climacteric; and, from that time onwards, instances are very common of progressive mental atrophy, with or without sensory and motor impairments. Though we find it expedient to consider "Consecutive Dementia" separately, those cases of acute idiopathic insanity, followed by dementia instead of recovery, are probably cases in point, and their acute attack merely an incident in the atrophic process. Some of them, after an indefinite period of dementia, undergo a more or less rapid motor paralysis from diffuse degenerative changes. Americans, under the name "Hebephrenia," describe a disease which occurs at or soon after puberty, which may include an acute attack of insanity, which leads to craziness and dementia in about a year. It occurs in lawless patients, and masturbation is a frequent factor. It would therefore appear to be an abrupt progressive dementia in the obviously degenerate.

Abnormal Mental Variations. — Alienism suffers greatly from a defective nomenclature, and especially from the misappropriations of generic terms to a too narrow application. Thus, degeneracy, which ought to describe mental arrest in general, is used almost exclusively in reference to the extreme, criminal cases. Similarly, the term paranoia would have been most suitable for the designation of constitutionally disordered minds not actually insane, but is used almost exclusively to describe a delusional variety. We must therefore fall back upon a roundabout phrase — abnormal mental variations—to describe the symptoms of those odd minds which are of the insane diathesis. To gather our threads together, we may recall summarily the most important wants of such minds. These are a lack of life-preserving instincts; defective sexual desire of a normal form; domestic indifference; lack of capacity for enjoyment; muscular ineptness; gullibility; unpracticableness; and tactlessness. These are the negative signs of the insane diathesis, not all of them to be looked for in one case. And associated with them we must note many positive symptoms—disproportionate developments of function which add to the mental asymmetry. Their name is legion, and we must confine ourselves to a few of the most important.

Of the primitive functions, the sexual claims first attention. History teems with instances of redundant sexual capacity of a normal form. And more significant are the various perversions of it, so hard to explain except as due to disarrangements in embryo. Of the other instincts, all manner of excesses might be described, but other kinds of disorder are more significant. Very important, for example, are disorders of sensation. A visceral hypersensibility is often to be noted, and still oftener, an undue attention to stimuli of the special sense organs. Similarly abnormal minds may manifest undue muscular aptness and develop extraordinary movements. The best instances are those musical prodigies whose technique is so far in advance of their feeling for music. But it is on a higher plane that we find the best symptoms in point—in great feats of memory, in the tenacity of fixed ideas, in extravagant beliefs, in too subtle thinking, in unbridled imagination. Of the last, day-dreaming is very important, as visionary activities of that sort are often the prologues of delusion or of insane impulses. On the emotional side of mind we must take special note of fearfulness, that relic of the fugitive consciousness which expresses itself in an unfounded apprehension of impending evil, a tendency to expect an evil issue out of every experience, a habit of "supping sorrow with a long spoon." No special kind of emotion, however, is characteristic, but rather an undue intensity of whatever feeling the mind entertains. In volition, impulsiveness is the most common excess. In the ill-balanced mind promptings of an imperative kind, and which are not justifiable by the facts of experience, call for a control which is often lacking.

And lastly, extravagant conduct is characteristic—a manner and behaviour which is on the way to the ridiculous, or which sails very close to the wind and verges on the disreputable.

As I have said, these signs of abnormal variation may be multiplied indefinitely, and from what has necessarily been little more than a list of the more important, the physician may gather little that will be of use in practice. It remains, therefore, to refer to a few of the more frequent types of persons who manifest the insane diathesis; but always with the premise that even those who seem to conform most closely to such types are not to be set down as abnormal unless there is an abundance of contributory symptoms. But many of the symptoms which have been mentioned will be found in some of the following:—

There is a type of men and women who are the impersonation of timidity—shrinking, cringing, diffident, bashful—whose knees seem to give way on the slightest provocation, who are always afraid, but who may be all the same capable of great and even of heroic things. There is the sentimentalist who wallows in feeling, who cannot take a cool and reasonable view of anything,—pious perverts who translate everything into the terms of a silly religion which finds almost no expression in a dutiful life; devotees of æsthetics, who sacrifice everything to their sense of beauty; and other sensuous varieties. There is the amorous type of persons who take the opportunity, in season and out of season, to engage the affections of their promiscuous victims; feminine men who dote upon male beauty, masculine women who fight shy of men, the sanest of whom find expression in asserting the rights of their sex; and others who make a fetish of prudishness and "dwell with inverted gusto" on the evil of a feeling which, in its normal form, nature has denied to them. Of another form are the grievance-mongers, persons of an inveterate suspiciousness, always smelling a rat, ready to take offence, hopeless as wives or husbands or colleagues in office. There are the half-insane snobs, devotees to "good families," toadies, and sycophants; and the "affiliative" variety who, on the slenderest grounds, trace their relationship with all and sundry who can be of use to them.

Then there are the recognised paranoiacs, victims of disordered thinking, many of them more or less degenerate. The generic feature of them all depends upon a temperament which indisposes them to get on smoothly with their neighbours. The classical type is the querulous paranoiac, a variety of grievance-monger who airs his disputes in public, and supports his case with obviously deluded arguments, who can never take a decision against him, carries his case perhaps from one court to another, and who often has recourse in the end to a violent attack upon some personage of note in order to attract the attention of an indifferent public. In private life the tactless bore is almost as grievous a person, full of superficial ideas, inordinately pleased with his sickly wit, quite oblivious of contempt, not easily snubbed. Then there is the gull, the butt of all his acquaintances, good-natured often and often secretly vain, whom any one can impose upon, and who never learns from experience. And Paul Pry is another type, the curious paranoiac, whose nose is always in his neighbours' affairs, or who, in a less harmful sphere, buries himself in useless investigations. Then there are the unpersuadables, whose ideas are hopelessly fixed and who cannot see the force of argument, or, if they do, "are of the same opinion still"; and all the cranks and fanatics, "idea-fiends," as America knows them, who find in teetotalism, anarchy, socialism, or a land-tax, the solution of all the world's difficulties. Lastly, there are the more or less thoughtless persons whose conduct is disorderly. Of these, we may note the dare-devil, who does anything he cares without apparent regard to safety; casual paranoiacs who cannot appreciate the importance which men attach to their time and to their property, careless, haphazard, unpunctual people who break their engagements and do nothing thoroughly or exactly; gushing men and women, voluble in expression, silly and empty-headed, except for a store of useless information; the would-be reformers who never see anything they cannot improve upon; and the feckless, who can never arrange their affairs, who spend their days in vain efforts to redeem lost time, always busy and never doing much.

These people, ill-nourished, or prone to excess, or careless of health, often come to an unhappy end, perish in some hopeless cause, pamper themselves with the connivance of foolish relatives into chronic hypochondria, or, driven from pillar to post, from post to pillar, end in an asylum. A little care and systematic inquiry easily furnish the facts by which to recognise the more precarious of these variations. We must not, however, conclude hastily that a man is of insane predisposition merely because of some conspicuous eccentricity or bad habit. From such judgments has arisen the preposterous fallacy that geniuses are usually degenerate. As has been noted, some abnormal people are prodigiously accomplished in certain directions. But the common sense of mankind revolts against the judgment which classes with them the really great minds of history. A Shakespeare, a Goethe, a Napoleon, does not manifest arrest of development, and a woman must needs have suffered much from modern teaching to be afraid to have such an one as the father of her children. These great minds apprehend much that is beyond the common perception, but their genius was based upon a strong fabric of primitive human instinct.

Other kinds of genius, still not necessarily degenerate, are instances of extraordinary developments of some special functions of the intelligence, such as the inventive, which do not imply an all-round sufficiency. Such minds *may* manifest an arrest of one or more of the important mental functions, and in that case insanity is more likely to overtake them. Physiologically speaking, genius is simply an unusual vitality of nervous mechanisms whose function is such as fits with the environment of the time. The same kind of account must be given of "strong men" (muscular geniuses) as of a great poet. But the sane genius is one whose extraordinary gift is the accomplishment of a mind which is fit for all the essential functions, and apprehends all the essential traditions, of his race.

Further allusion to this subject will be found in Dr. Macpherson's article. For these odd minds are apt to end in delusion, or to suffer that combination of degenerate feeling and degenerate thinking which constitutes Paranoia (*q.v.*).

These abnormal variations which we have considered call for prophylactic treatment such as Dr. Urquhart has suggested, and a great future lies before mental science in this application of its knowledge. But it will not be until the public realise that the brain is an organ capable of scientific management, and not until parents and practitioners habitually observe and study mental facts, that we shall enter into this, the only hopeful field, of mental medicine.

IV. The Incidence of Episodic Insanity, with Special Reference to Bodily Signs

As an episode in the life of man or woman, whether of the insane diathesis or no, insanity is nearly always ushered in by characteristic signs, and there is no aspect of the subject of greater importance to the physician. Again and again he will be face to face with some one who is in very bad spirits, or unduly exalted, or expressing some strange beliefs. Is this person then merely depressed or joyous from some passing cause, and are these strange beliefs merely mistaken but intelligible opinions? Or is there here the incidence of an insanity—the invasion of the organs of personality by a disease which may or may not be checked, but which in any case is sure to be grave? And these questions should be promptly answered, for a large majority of the insane are curable, and early diagnosis is of the first importance.

Speaking generally, nearly every case of insanity is attended by a more or less profound disturbance of all the important bodily functions; and the more grave the insanity is going to be, the greater that disturbance is. Before acute insanity the following changes are to be noted:—

Nervous System.—Disturbance of sleep is very common, either as sleeplessness or as dreams, much more rarely as extreme somnolence. In melancholic insanities anergia is usual—a disinclination for exertion and an oppressive lassitude. In mania and in delusional insanities the patient has often manifested restless energy. The muscular system is generally irritable, and tremors, slight spasms, and exaggerated muscle reflexes are almost invariable. A great number of cases complain of hyperæsthesia.

Digestive System.—Digestion is almost invariably disturbed, and loss of appetite is nearly constant in melancholia. Dyspepsia is associated with constipation, which the patient is apt to ignore, and the tongue indicates it.

Circulation.—In every case in which melancholia or mania is imminent a change in the character of the circulation occurs, but there is not always a characteristic change. In both diseases the pulse begins to be variable and excitable and fickle in rhythm. There may be hypertension or hypotension in either disease, just as in sane joy or grief. The important point is, as Dumas proved (*Revue Philosophique*, June, July, August, 1896), that the circulatory change always precedes the mental. He describes a case of alternating insanity in which he was able to predict the attacks from the pulse. There may or may not be stasis and congestion. Œdema and asphyxia of the extremities is common in lethargic melancholias and in stupor.

Changes in the constitution of the blood have often been observed, but the practitioner is not likely to know what any given case normally manifests in this connection.

Urine.—We may say of the secretion of urine, as of the circulation, that when insanity is impending there is usually a marked change, but that the change is not of a distinctive kind. Perhaps the commonest fact about patients before becoming insane is that, instead of an equable secretion, they suffer from a diminished quantity, with high colour and high specific gravity, with periods of polyurea in which the urine is pale and of low specific gravity.

General Constitutional Changes.—These are most important. In a large majority of cases the patient has been losing weight. Very rarely is the incidence of insanity coincident with increasing weight. There are two great types of constitutional change to be noted. In one set of cases the patient is observed by the relatives to be growing thinner, paler, more wasted and haggard in appearance. In another there is an appearance with which I always associate the word "surfeited." To the eyes of relatives the patient appears more gross—not necessarily gaining weight. The complexion becomes muddy, the expression heavy, the eye dull, and the patient himself may complain of feeling "clogged up." Perspiration is usually very scanty, but the skin may be greasy. This appearance of surfeit is unmistakable, indicates a defect of excretion, and probably denotes toxæmia.

Reproductive System.—Menstruation is almost invariably disturbed before insanity and the sexual function modified.

The *mental signs* of impending insanity need not be enlarged upon. However, it may be noted that, except in the rare cases of almost instantaneous onset, the patient's habits invariably change before an attack; the domestic, personal, and business traditions are departed from. That is much more significant than any actual feeling or idea expressed by the patient. If the state of mind which excites the physician's suspicion has not been preceded by altered habits, there is almost certainly no acute attack impending unless there are some extraordinary circumstances to cause it, a disaster or other great shock. We must therefore inquire carefully into the circumstantial facts in every doubtful case. In an interview with the patient greater importance attaches to facial expression and to manner than to what he says and does. Before insanity has become actual, patients almost invariably change in appearance, and express preoccupation, suspicion, or dread, even if they do not admit it. At the same time the face assumes an unwonted brightness or dulness.

In every instance it is a change from what is normal in the patient's case that is significant. Nearly every function which I have specified—both mental and bodily—will be found to alter when acute insanity is coming on, and the physician who systematically investigates his case will very rarely be taken by surprise. When the attack has begun, and during its course, these bodily symptoms become aggravated. Derangements of a constitutional kind and disorders of one or other of the systems are almost invariable in episodic insanity. But the symptoms are so variable that we shall not enlarge upon them in the following account.

V. Types of Episodic Insanity

Having glanced at the permanent abnormalities, we now consider acute insanity as it appears as an episode in the life-history of a mind. Sometimes these actual insanities occur in persons such as have been described as of the insane diathesis, and in that case the disease may be traced as a gradual development of habitual modes of thought and of feeling. In other words, a constitutionally abnormal personality may become more abnormal and cross the line beyond which the patient may not be regarded as responsible or as fit to take charge of his life and affairs. Or some accessory cause or some crisis in development may lead to a gross cerebral disturbance, which becomes evident as a definite and well-marked departure from the usual mode of consciousness. And that is what always happens in the case of persons who, though not of a recognised type of abnormal mental variation, suffer an episodic insanity. From any of the causes, except inheritance, such as Dr. Urquhart has enumerated, a fairly orderly mind may suffer a cerebral attack, and such an insanity is an invasion of the personality, slow or sudden, under which the normal character may disappear and give place to feelings and beliefs and conduct which are quite alien to it. That is, subconsciousness may usurp personality. And it must greatly enlarge our conception of the subconscious to study types of insanity. For the very fact that there are such types implies that the human brain, on a plane a little lower than that which becomes realised in sane life, is still a more or less constant and systematised organ. To my mind that is a very important conception, and one which is perhaps not realised by all physicians who concern themselves with mental functions. In a lesion of the motor area with partial paralysis the abeyance of movement is not followed by indefinite incoördinations, but by secondary symptoms which are systematic. If the lesion is unilateral, muscles of the sound side produce an excessive effect, as in pushing the tongue or drawing the mouth aside. Similarly, if the lesion is high up, the descending order follows quite definite and systematic paths. The spastic gait or that of tabes is characteristic because it is more or less constant, because the excessive contractions in the surviving muscles depend on the systematic arrangement of the surviving mechanisms. An exactly analogous system or organisation pervades the lower mental areas, so that, when personality fails, the brain does not manifest sheer disorder, but usually reveals a typical activity of surviving mechanisms. What type it will be depends upon constitution and upon the seat of the lesion. The best example in point is that of alternating personality (double consciousness), a most interesting and not uncommon form of insanity. In that disease, when the first personality subsides, the cerebral activities are determined by mechanisms of a second personality which were, so to speak, waiting in readiness for their opportunity. But though not so obviously, every insanity reveals an analogous orderliness. Even acute mania is not really a disordered but a quite systematic over-activity of sensori-motor mechanisms. Were these facts not so there would be, as I have said, no types of insanity, and the insane would be perhaps weeping because they were millionaires, or committing suicide in order to get the crops in, or making homicidal attacks on all and sundry because tables should be made of glass. Such real incoherence is extremely rare. Moreover, the types of insanity which we do recognise are for the most part secondary symptoms. They are analogous to a description of motor paralysis in terms of the contractures and hyperæsthesias of the surviving mechanisms, as Hughlings Jackson long ago pointed out. Acute insanity is a greater or less degree of

failure of normal personality with all that that entails. The primary lesion is in the mechanisms of sane character and sane conduct which have gone by the board, leaving an activity in lower mechanisms by which we designate the disease.

It has been suggested by many authorities —Bevan Lewis, for example—that clinical varieties of insanity are not to be regarded as essential, that they are "purely artificial or arbitrary divisions," that the pathological process, vascular or nervous, is the important thing. The practitioner, however, wants first to know what the clinical developments in any given case are likely to be, and he is right in insisting upon that knowledge. For in all nervous affections the site of the lesion is as important as the lesion. It is important for our knowledge of disease to distinguish between thrombosis and glioma, between apoplexy, embolism, and sclerosis. But in each case the consequent impairments are part of the illness, and what they will be depends upon the site and size as well as upon the nature of the lesion. And these things are still more true of the lesions of insanity. We may safely defy any one to predict the differences in the course of a mental disease which is due to slow vascular obliteration, and one which is of the nature of a slow neural atrophy. The differences that we recognise are chiefly differences in the positive secondary symptoms. Abeyance of normal personality is the prime fact in all episodic insanities. Thereafter the course of the disease depends upon what groups of lower functions become too active, and that is an affair of anatomy—a topical factor. That experiment and disease have localised and demonstrated the Rolandic centres does not make them more real. Hughlings Jackson and others knew of their existence long before. And similarly, recognising as we must a domestic mode of consciousness, for example, a fugitive, an amorous, a constructive, we are bound to infer a systematic mechanism for them; and our knowledge of it will not be greatly enhanced even if, as seems unlikely, some one should demonstrate their position and form. There are exceptional cases, but the vast majority of insanities are only intelligible as systematic secondary affections. Were consciousness and its mechanism not systematic there would be, as I have said, no clinical types of insanity.

Melancholia.—Melancholia is characterised by an abeyance of interest in things in general. That is the primary fact, the *negative* symptom. Following that loss of habitual conduct and of interest, the patient becomes self-absorbed, dwells upon his misery, seeks to find some explanation for it, or perhaps tries to put an end to it by insane violence. In any case he cannot forget his pain, and that self-regarding mode of consciousness is the secondary, the *positive*, fact in his case, and it is constantly present in melancholia. Moreover, this mode of consciousness is not fortuitous. It is determined by subconscious system. Self-regard to an insane degree is provided for by inherited cerebral mechanisms, and would predominate in all human minds but that higher interests and activities overrule and prevent it. Melancholia is the simplest, the most likely result of insanity. Its primary features are, as I have said, a loss of physical energy, an anergia, so that the patient is not fit for usual activities, and a loss of interest in life, so that the mind does not attend to affairs. A very slight investigation will reveal these facts. A melancholic is habitually tired, and if compelled to undergo great exertion will certainly become worse, and probably proceed to mania or to dementia. But the exhaustion is most notable in the highest functions, such as are normally concerned with the conduct of life. Hence, as Dr. Mercier points out (p. 507), the direct benefit of a complete relinquishing of all responsibilities and duties. In many cases the nervous depression is due to bodily disorder, but at the same time the failure of the functions of personality induces or occasions physical deterioration, and we can never exactly say how much of the depression of bodily functions is a cause and how much an effect. The fact remains that a little inquiry will reveal disorder in nearly every system—dyspepsia, constipation, and anorexia, a variable heart, a soft pulse, cold, and perhaps swollen extremities, diminished secretion of urine, dry skin and hair, a loss of weight, sleeplessness, muscular irritability and flabbiness, diminished surface sensibility, exaggeration of deep reflexes, and often neuralgia, stiffness of muscles, and difficulty and slowness in executing fine movements.

The loss of outward relation is the central and primary impairment of mental function—the abeyance of object consciousness, as Bevan Lewis designated it. The melancholic is interested in nothing and in nobody in his neighbourhood. He neglects duties and pleasures alike, and forsakes his habitual occupations. When the disease is advanced even striking events pass almost unnoticed, and emergencies induce no response. That of course betokens a serious failure of personality. Thereupon lower forms of nervous activity become apparent, chief of which is the self-regarding mode of attention—the rise of subject consciousness. That is one definite phase of subconsciousness. The mind, on relinquishing usual affairs, might become occupied with the unusual, as on a holiday, or in delusion, or in mania. In melancholia it becomes occupied with itself; and a persistence of that form of consciousness is inevitably associated with unhappiness. Pleasure

is the content (the subjective fact) of activity for which there is ample cerebral energy. The conditions of pain are inactivity or activity in excess of vitality, both of which are present in melancholia. The exhaustion from which the melancholic suffers occasions weariness and distress, and his inward regard perpetuates it. All the rest of melancholia is the attempt of the brain to give expression to its altered activities. It is probably a misuse of terms to speak of the melancholic personality, for when the old personality has gone—we cannot say whither—what remains is not worthy of the name, but we may call it the melancholic or inward character. And that expresses itself in various ways which determine what are somewhat arbitrarily discriminated as varieties.

Simple Melancholia.—In *simple melancholia*, for example, outward movement is of the slightest. The patient is simply and monotonously depressed and quiet, does not moan much, or otherwise express grief. The intelligence may be fairly active and the patient able to appear in society without attracting attention, though always with a burdened mind. When alone, however, he probably resigns himself to his pain, and already has glimmerings of delusions such as might seem to him to account for his distressed state. The characteristic fact of this mode of consciousness is this constant undercurrent of pain—a disorder exactly analogous to exhaustion of any peripheral part, though, for reasons referred to in the account of the mind's relation to the brain, the patient does not refer his pain to his cerebral mechanisms, does not locate it. The condition is what is known as a systemic sensibility of an unpleasant tone. In health the sum of cerebral activities has a balance of energy on the right side, in consequence of which the systemic sense is pleasurable, and creates a joy in life. The melancholic, on the other hand, does not find it good to be alive, and from a very early stage would not be sorry to die, and may even feel promptings to end his misery by suicide. And that undercurrent of ill feeling can nearly always be diagnosed, even if the patient will not plead guilty to it. If you watch him unobserved, you will see him assume that indescribable look of self-absorption, as, perhaps, in the middle of some occupation he turns his mind inwards with a sigh, and from vacancy his expression will pass to one of actual pain. Moreover, melancholia has what is called a gymnastic—a definite muscular state, just as exaltation has. Over and above the slight degree of atonicity which prevails, and which occasions attitudes and movements that betoken slight muscular feebleness, the voluntary muscles assume positions of automatic contraction. The characteristic pose of exaltation is in general one of extension. But you will rarely observe extension in a melancholic unless he is making an effort. When he lies down his legs automatically flex, and his arms and hands. Similarly his brow puckers downwards, his mouth purses, and his eyes are rarely wide open. I am speaking, of course, of what happens when the patient is not aware of his movements. In other words, the muscles are beginning to assume the poses appropriate to grief, and which in their extreme form are known to us all as revealed by the actor's art, or as commonly expressed in sane woe. Other varieties of melancholia similarly suggest that the disordered activity follows a systematic course.

Acute Melancholia.—The progress of disease from simple to acute melancholia is obviously systematic. It is in fact merely a deeper degree of melancholia. The outward regard, occasionally possible in the simple variety, is now wholly impossible. Self-regard correspondingly increases, and so also does its accompanying pain. Similarly the gymnastic of melancholia is intensified, and the patient's muscles automatically assume poses and attitudes which are stereotyped as well-marked expressions of distress. (Note that, except on the stage, these shows of grief are rarely observed in the sane.)

In acute melancholia volition is profoundly affected, so that the patient is no longer able to conceal his grief, and with every new loss of power self-regard is accentuated and its consequent pain. Or to express it more correctly, outward regard is still further lost, and nothing in the environment has its proper influence on the consciousness of the patient. In many cases this is so pronounced that even if you speak to him, or try to move him about, he behaves as one in a painful dream, not looking up, not speaking, apparently never turning his attention from his distress. And it is important to observe that this excess of self-regard is without adequate reference to normal bodily facts. For while in the visceral variety of melancholia there is over-attention to internal stimuli, the consciousness, in simple and in acute melancholia, does not sufficiently embrace ordinary physical facts. Attention to what we may in a very wide sense describe as the "calls of nature" are inhibited. The melancholic seems not to be aware of the discomfort of an empty stomach, of a full bladder, of drenched garments, of an unwashed and unkempt person, or even of minor surgical injuries. And much of our treatment must be a provision against the dangers of such a state. The motor signs are similarly accentuated, and the classical expressions of pain sit, from morning to night, on the patient's face, hands, shoulders, trunk, and legs. And now also the morbid activities find their way in systematic directions much more obviously. There is much more mental movement of a disordered kind. For example, instead of the intervals of sane thinking of which the simple melancholic is

capable, and which alternate with perhaps simple attention to pain or vague surmises as to its cause, the patient becomes possessed with an emotion which he explains by means of delusion. And that emotion is nearly always to be described as fear. Acute melancholia reveals the survival of the fugitive consciousness. The patient is pursued by fear, very often in the form of remorse, terror at the thought of the possible consequences of the wickedness or folly of which he imagines himself to have been guilty; or his panic takes a prospective direction, and he is constantly expecting a dreadful misfortune; or he believes—which is most likely in those cases in which there is some ground for it, and which is occasioned by visceral sensations—that he suffers from incurable disease. Now also the disorder may be observed to spread towards the special senses, and attention, finding no foothold in sane and outward activities, becomes possessed by suggestions which emanate from the visual areas, or more often from the auditory. In the beginning this is evident merely as hyperæsthesia. The patient hears far too much. Or, perhaps, we would express it better as over-attention and say that the patient listens far too much. And that happens which we might expect if the disease is not stayed. Watching and listening for sights and sounds instead of attending to affairs, the patient will become the subject of hallucinations, at first vague and irrelevant, but later appropriate to his distress. For here, again, we have to note the systematisation of the disorder. We cannot suppose that in this state of disordered perceptual activity only appropriate hallucinations occur. But the distressed attention selects what is relevant, and, as always, attention to the distressful increases the facility of their occurrence; and so painful hallucinations survive over others. All these are the positive survivals of activity in acute melancholia. In this condition purposeful acts are almost in abeyance. But there is still the risk of suicide of an impulsive kind, though the patient is now unable to plan and to scheme for it.

Acute Delirious Melancholia.—Acute delirious insanity generally takes the form of mania, but not always. As has been hinted, the prime difference between mania and melancholia is the survival of outward activities in the former. In acute delirious melancholia these movements are not found, but other symptoms of it are present. In some cases of acute melancholia the signs of bodily disorder become very prominent. In particular, some patients take on what I have described as the bodily signs of surfeit so suggestive of toxæmia. And that may go on to an extreme state in which, with diminished or altered secretions, furred tongue, sordes about the mouth, dry skin, rapid pulse, rise of temperature, and prostration, there is the apparent unconsciousness of surroundings and of the self which is characteristic of delirium. In this stage then the primary, *negative*, fact—the lostness of the sane personality—is more than ever obvious. So also the secondary, the *positive*, symptoms are on an ever lower level of cerebration. The patient has ceased to be simply self-regarding and distressful. His conduct, and presumably his consciousness, are of the delirious mode, as in typhoid or in pneumonia. Even the gymnastic of pain has gone, and the patient's muscles move automatically in delirious movements—chiefly muttering and picking. In all probability the consciousness now is barely capable of description. It would appear to be occupied by flitting sensations and by quite vague and dream-like ideas. The over-activities with which it is engaged are apparently in the Rolandic area or below it—unrelated disturbances of motor and sensory mechanisms of a primitive form.

Delusional Melancholia.—Simple melancholia, if not cured, may go on to an acute form: or the acute or the delirious variety may develop very suddenly without passing through that stage in which the depression is a background to a life still capable of outward activities. On the way to becoming chronic, nearly all melancholias are delusional. Such a development is probably less painful; for, obviously, when the patient is constructing an argument to explain his state, he is not entirely self-regarding. Delusions are, of course, *positive symptoms*—the wandering fancies of a "lost mind," which betoken activity of a secondary kind in the now-surviving mechanisms of intelligence. And here, again, we must note that the melancholic mind abides by fairly constant paths. Nearly all melancholic delusions refer to one or other of the primitive instincts or traditions of the race; and, having taken a line, each patient generally abides by it for a considerable period. It is always a good sign when the subject of the delusions alters. When the consciousness departs from one set of delusions it is quite likely to be on the way towards recovery. The most usual types are as follows:—

Consciousness of Immorality.—Many patients are convinced that their distress is the consequence of their evil deeds. And not infrequently there is some truth in the idea. A large number of patients who believe that they are eternally damned have been the victims of a vicious habit, and a bad conscience is a potent cause of insanity. A considerable number of very righteous persons, however, believe when insane that they have been horribly wicked. As a rule the belief is vague. But sometimes the patient conceives a concrete crime—murder, or infidelity, or theft—and harbours the delusional belief that he has been guilty of it.

Consciousness of Persecution.—Delusions appropriate to a fugitive consciousness characterise a form of slow insanity which we shall consider

later. But many melancholics also, bowed down with pain, conceive the idea that they are the victims of a conspiracy, or wanted by the police, or pursued by an angry God, or played upon by electricity, or undergoing slow poisoning. These delusions in such cases are not abiding.

Consciousness of Social Disaster. — Many patients on retiring from society under their grief become possessed with the idea that they are disgraced in the eyes of their neighbours, that people look askance at them, that they are defaulters and ruined. Melancholics who seldom manifest an interest in outside affairs will sometimes devote themselves to the newspaper to an extent difficult to understand until we learn that they are looking to find themselves in one of its black lists. Similarly, when a patient is removed from home a very common delusion is that a domestic disaster has occurred, that the home has been burned down, that the spouse is dead or has fled, or that the children have been murdered.

Consciousness of Bodily Disorder. — These classes of delusion are not discrete, but obviously overlap. No description of mental facts is or can be exclusive. And many delusions, otherwise classified, are occasioned by a consciousness of ill-health, whether interpreted as such or described in other terms. That mode of attention is sometimes general—does not refer to any particular function or part of the body, and then the patient probably believes simply that he is dying, or labouring under some incurable unspecified disease, or emitting an offensive odour. Or it may be particular—refers to some one system or symptom—and then perhaps the patient believes that insects are crawling over him, that his stomach is of glass, that he has cancer of the throat, that he has lung, or heart, or abdominal disease. The systemic ill feeling such as occurs in neurasthenia or in toxæmia probably accounts for this hypochondria of a general kind. And we call it hypochondria, not because there is not actual disease, but because the patient suffers the invariable hypochondriacal symptom of being wholly unable to ignore his trouble, and, not content with feeling and thinking about it, discourses freely upon the topic. Particular delusions are more often based upon local disorder. Hyperæsthesias and anæsthesias probably account for many such beliefs. Visceral delusions often prove to be approximately true, and physicians do well to make frequent examinations of patients who insist that a specified function is disordered. In particular, as Dr. Macpherson, in this country, and others have recently insisted, we must have great regard to indigestion or paradigestion in the insane. The belief of a melancholic that he has an animal, an embryo, a clock, or a battery "in his inside," is often a misinterpretation of a very real fact. A large proportion of melancholics are dyspeptics and have dilated stomachs. Many hours after a meal, if vomiting occur, or if the stomach be emptied by the tube, undigested food will be ejected. Hence the modification of the old idea that melancholics should be indiscriminately stuffed with food, and the advantages in certain cases of washing out the stomach, and, as Dr. Mercier suggests, of a strictly regulated diet.

Suicidal Melancholia. — If melancholia occurs, as it often does, in a person who is lacking in the life-preserving instincts, he will certainly become suicidal if his distress is considerable. We can appreciate what it means to him if we consider (what is a fact and not merely an expression) that a victim of a bad sea-sickness really does not care whether the boat goes to the bottom and he with it or not. The distress of melancholia, and probably of stupor, is what we would describe as intolerable. Most cases would be glad to die, but most cannot bring themselves to perform the suicidal act. The prolongation of life is very largely a muscular affair. The life-instinct is to a large extent an inherited series of self-defensive movements—to draw a limb away from hurt, to run from danger, to shrink from inflicting a wound on oneself—all of them as automatic as the blinking of the eyelids in a dust-storm. In suicidal melancholia one of two elements, or both, may be present. These instincts for danger, the common-sense appreciation of what will hurt, may be not fully developed, or the patient may have great pluck or determination, hence the discrimination which patients show as to the mode of suicide, and the discrimination which the physician must exercise as to the fatal probability in a given case. The only safe assumption is that each and every melancholic will be glad to die, and therefore it is never safe to leave them unguarded in circumstances which offer, and perhaps obtrude, an opportunity of suicide. But the variety of melancholia which we are now considering is a much more positive state. It is not merely carelessness of life, but a fixed and persistent determination to put an end to it. Such cases are not infrequent. They are not of the acute form of melancholia, which has almost no outward relation, but generally speaking have considerable mental activity, probably converse a good deal with their neighbours, and are able at times to quite conceal or forget their distress. Their state of mind is like that of simple melancholia with a suicidal intention added. They are like persons who know they must die soon, sometimes are cast down by the thought of it, sometimes mercifully forgetful, sometimes deliberately deceitful about it. Every such case has an insane conviction on the subject—that his or her death is necessary or expedient. Sometimes that conviction grows in a mind which does not reveal much other insanity, but quite as often is an insane conclusion from insane premises. Such patients believe, for example, that they are a source of danger to

their neighbours, that the world will not go right till they are dead, or that they must die to expiate their crimes, and not infrequently the grounds for bringing about their death are furnished by hallucinations. From that of the religious fanatic who hears God calling upon him to come away from the earth, to that of the degenerate criminal who is beset from morning to night by fierce voices which cry out upon him that he must be slain, there are endless forms of hallucinations such as may promote the suicidal determination.

In suicidal melancholia, then, the systematic nature of the disease is obvious. It probably occurs in people who have not a normal fear of injury and of death. This lack of life-preserving instinct is an inherited quality of a negative kind, and when useful activities of an outward kind become impossible and pain supervenes the consciousness assumes the suicidal mode. It is fortunately rare, for all pronounced melancholics would be suicidal were it not that the vitality of the life-preserving instincts is enough to withstand and inhibit suicidal suggestion. But they all suffer some degree of abeyance of these instincts in their disregard of the calls of nature to which I have referred. No acute melancholic takes pains to live, though only a few take pains to die.

Homicidal Melancholia.—Just as the melancholic's distress may find a systematic expression in a determination to commit suicide, so also may it find an outlet in homicide. Here again the apparently disorderly activity is one which has a definite place in the patient's consciousness. Just as he might believe his own death important, and bring it about, so also he may come to think that some one else should die, and attempt to achieve that. Sometimes these suicidal and homicidal intentions are associated. Hence those tragedies in which a father kills his family and then perhaps himself. In some cases the homicidal melancholic entertains a delusional belief about people in general, such as that the more people he can kill the better God will be pleased. Sometimes he believes himself called upon to extirpate a class of persons—the profligate perhaps, or the oppressive—and in his weak thinking he may attack persons who are not of the class referred to. More often the grounds of homicide are personal, and the patient attacks persons whom he believes to be in conspiracy against him or otherwise the medium of his misery. And then also the signs by which he recognises those whom he considers his enemies are generally fanciful and slender. Lastly, he may select one individual, whom he may delusionally endow with evil powers, and plot and plan for his or her destruction. And in such cases we have specially to protect such persons towards whom the patient has conceived an antipathy. Often because of some real slight or injury, sometimes from unreasoning dislike, the patient will develop an extreme vindictiveness as of an elephant or dog, supporting it with delusional arguments, and seeking an opportunity of homicide. To such cases the remarks offered as to the constitutional predisposition to suicide may be applied also to homicide, but translated into terms of the others'-life-preserving instincts. The instinct not to hurt or kill others is strong in our race, or if not always so, is carefully educated in most people.

Agitated or Expressive Melancholia.—These varieties of melancholia which I have indicated are obviously a transition of the disorder downwards and outwards. The morbid state in simple melancholia is centralised. In delusion, when the mind thinks about itself and explains itself, the emotional content is diminishing; in suicide there is a distinct though insane activity of a purposeful kind; in homicide it is still less central, more outward. And in the variety called agitated melancholia the outward activities, though on a low level, are very manifest. The patient wrings her hands, tears her hair, sways up and down on her chair, or perhaps scurries about the room like a frightened rabbit, moans, groans, or cries aloud. In short the condition is one in which the gymnastic of distress in its most extravagant form has supplanted the distress. The consciousness has become a motor consciousness, and probably very little pain is felt. Actual shedding of tears is uncommon, and, moreover, when the patient is left to herself, the agitation often diminishes, and she is capable of apparently sane conduct. It is in relation to the presence of onlookers, or the knowledge that people are near, that is the occasion of agitated melancholia. In that respect the muscular activities of agitated melancholia differ from those of mania. Maniacal acts do not always express joy, are often without reference to the central state. Melancholic acts usually express grief, are the recognised medium of expression of the distressed consciousness. But angry mania and excited melancholia are not far removed.

Any attempt to interrupt these movements of grief or of fear is usually resented, and the muscular pose becomes one of resistance. The consciousness of fear begets an active distrust; or at least the conduct of a resistive melancholic seems to imply that. But we err in assuming, in the insane, a mental content appropriate to their conduct. Their consciousness probably does not contain what the muscles express, and perhaps resistiveness is merely a muscular reaction implying an unthinking aversion to interference. These resistive cases are most difficult and call for most skilful nursing. The worst of them stiffen on the very approach of doctor or nurse, cling tight to the chair if

you try to raise them, root themselves to the ground if you try to make them walk, clinch their teeth if you would feed them, refuse to swallow, to wash, to dress, to undress, or even to empty bowels and bladder. Further, they often kick and bite, and scratch and pull the nurse's hair. They call for immense patience, and people are apt to regard them as wilfully disagreeable. That is because of the outwardness and apparent purposefulness of the symptoms. Even the most uninitiated recognise that the quiet melancholic is irresponsible. But the resistive melancholic seems responsible. Sometimes we may help the nurse by reminding her how a trapped animal, or a child in distress, will repel and perhaps attack those who go to its help, and that in resistive insanity the patient does not understand people. Chronic melancholia, which may persist for years, is usually of an active form and often resistive.

Phobia (*Partial Melancholia*).—Many cases, and some of them very interesting, without a general melancholia, suffer a morbid and persistent fear in relation to limited and specific things—knives and scissors, blood, and blood-red colour, narrow places (claustrophobia), open spaces (agoraphobia), and what not. Some of these phobias are developmental, and the common mind is normally afraid of gross darkness, loud noises, dead human bodies, reptiles, precipices, fire, deep water, and other things properly to be avoided. In the weak, either by development or after critical bodily illnesses, or in other neurasthenic states, the affection often relates to things not essentially harmful. The fear, whatever it may refer to, primarily occurs only when the patient is face to face with the object of it. But in many cases of a melancholic habit the patient dwells upon the fear, and, apart from any objective occasion, becomes distressed at the mere idea of the thing. Then very probably obsession will develop, in which the nauseous suggestion obtrudes itself repeatedly and persistently until the distracted patient breaks down under it. In any case, while the terror is upon him the patient is in great distress. And the panic which rivets his attention is associated with a characteristic muscular state, not unlike the gymnastic of acute melancholia, but with an added tremulousness and an accentuated feebleness. At the same time the pupils often dilate, the face turns pale, and the patient feels himself unable to move. In fact, the differences and resemblances, mental and bodily, between melancholia and phobia are analogous to those between unhappiness and fright. Moreover the treatment of phobia practically resolves itself into preventing its becoming melancholia,—by diminishing the general, and diverting from the particular, nervousness from which the patient suffers.

Stupor

When insanity is described as a disease in the cerebral structures which subserve normal life, we imply that the function of nerve-cells and nerve-fibres is impaired in the sense that their excitability is diminished. Such an abeyance of functional activity becomes manifest, when it is far advanced, in changes which we can observe under the microscope, such as are described by Dr. Robertson. But, following that impairment of excitability in the cells and fibres of sane mental activity, there is usually very evident in insanity a hyperexcitability of mechanisms on a lower level, or, at least, an abnormal activity in them, and these secondary excesses constitute the positive symptoms of insanity. But stupor must be described almost entirely in negative terms. It is characterised by failures and losses rather than by excesses and extravagances. The patient, in pure stupor, cannot do anything except the most primitive things—cannot speak, cannot walk, cannot shake hands, cannot brush his hair, cannot button, and, presumably, cannot think. In other words, and to describe the disease in a sentence: Stupor is the cessation of conduct. But, before elaborating that remark, we must digress to consider exactly the system of functions which we must suppose are chiefly involved in the diminished excitability which characterises the disease.

In his description of melancholia, Bevan Lewis lays much emphasis upon the failures of the *muscular element of mind*, and indeed regards them as primary. The subject, however, is one which is perhaps more apposite to stupor. And that is in no way at variance with Bevan Lewis's position. For stupor is a secondary development of melancholia in which the depression of function, the increased neural resistance, has spread downwards and outwards towards the muscles. Indeed we might have described a stuporose variety of melancholia as leading us on to stupor. There is no case of acute melancholia that does not threaten stupor; and there is no stupor that is not, or has not been, melancholic.

The term "muscular elements" as applied to intelligence is easily understood. Based upon the muscular sense, it has its final expression in the constructive or inventive consciousness—in conceiving and designing things to be done and made. Its most elementary form is necessary to all perception. The shape, the size, the distance, the feel, the weight, the direction in which things are—all these and other qualities of external objects are perceived by muscular sensations following movements of the eye, limbs, and body. Bevan Lewis thinks that these muscular elements in the melancholic's perception of things may be impaired before and without an impairment of the other and

passive elements of the senses, that the "space attributes" of external objects can only be realised by an unwonted effort, and that thereby an excessive sense of the "resistance of the environment" is suggested, "and the apparent energy and freedom of the will restricted." (We may doubt that, seeing that the elementary qualities of things are not known to the adult mind as discrete, but as compounded, and in course of dissolution, the compound mechanism of a given object into which its primal elements have been resolved will be the first to go.) Be that as it may, there can be no doubt of the importance of another form of the muscular element in intelligence—what we may call *projective activities.* These are not relations of what is commonly recognised as sensory activities, but are on the other side of mind. They are functions of what we know as psycho-motor mechanisms, and might be described as aspects of the mind in the act of moving. These are not only muscular movements, but mental movements as well—the forward activities of the intelligence such as occur in reasoning, deciding, choosing, planning. For much more of the mental cerebrum is psycho-motor than merely that part of it which contains the images of definite movements. The idea, the intention, the mental picture of any definite movement which we may project, is well down on the way to actual motor functions. But many of the more central processes are of this projective nature. For example, most people think about things, not by making use of mechanisms which represent conceptions of the things themselves, but of those which represent their names. And names are things which we speak or write, or which our eyes pick up from the printed page, or which we prick our mind's ear to listen to—all of them muscular elements. Many other mental processes we can describe in terms borrowed from the analogy of gross movements, and which suggest their projective nature. The understanding "grasps" things (Bevan Lewis) and "holds" them; we re-"collect" things as the attention casts about to "pick up" what is wanted; we "pursue" a subject, "follow" or "take up" a line of thought; our sympathies "embrace" things and people; the imagination "reaches out" to "touch" new things; we "hold fast" to convictions; etc. etc. And we speak of a "standpoint" or "point of view" where the main self, as it were, remains while the projective activities go on around it. Above all, personality, as was said, is determined by what a man conceives of himself as saying and doing—the things which he consciously projects. All these then, and others, are the psycho-motor activities, the muscular elements, which fail in melancholia, and whose failure determines the abeyance of outward consciousness.

In stupor the psycho-motor loss is at its greatest, and the systematic course of the disorder is directly outward or downwards towards the muscles. Stupor is pre-eminently a psycho-motor paralysis involving even the mechanisms of definite movements of which I have spoken. The patient cannot reason, cannot decide, cannot plan, cannot arrange in his mind to do anything, nor agree within himself as to what is best to be done, or, at least, he gives no evidence of such mental movements, for, as we know him by actual observation, he is simply a person who cannot speak, cannot use his hands, cannot walk, can hardly stand, because, apparently, he cannot conceive of these movements. And by and by if the disease progresses, impairments on a still lower level will occur, and the patient becomes muscularly impotent, paretic, and atrophic.

By its nature a patient in stupor is prevented from revealing to us the mental content. He cannot express what he feels or thinks, or only in the most laboured fashion. This point must be insisted upon because some observers make very definite statements as to the actual condition of the stuporose mind, some of which are not verifiable. It has long been the custom, for instance, to regard stupor as a condition in which sensation is blunted. That may be so, but all that we can really say is that the patient does not react to the stimuli applied. And, from some necessarily rough experiments, I am disposed to think that, at least in some cases, there is not any gross loss of common sensation. Further, we cannot always take the subsequent version of the patient as descriptive of his content during the attack. We have no proof that memory is active during stupor. And yet we can infer a good deal with comparative assurance. We can confidently affirm, for example, that the stuporose state is essentially painful; we rarely see so pathetic an exhibition of profound distress as some convalescent stupors reveal. Both of the accepted conditions of pain are associated in stupor—a demand for response from enervated mechanisms, and pent-up activity in mechanisms not sufficiently used. Yet not in every case can the distress be observed, for even the gymnastic of pain may be lost, and the facial expression and the attitudes be those of almost pure anergia. The disease is to a large extent systematised in the highest motor mechanisms. It has no gymnastic in the proper sense, no characteristic muscular poses. The obvious thing in stuporose muscles is their absence of pose. The face, trunk, and limbs are expressionless and toneless.

Simple Anergic Stupor.—In this condition the patient can only be described in negative terms. It is one of the few insanities in which the obvious impairments of personality probably correspond to the actual disease. There is a profound disturbance of outward relations. The patient may, for all we know, feel an interest in

affairs, but he cannot show it. He just sits still, or lies still in bed, does not speak, does not *use* his limbs, although he may move, is fed and relieved of excreta only with difficulty, does not wash or clothe himself. The appearance is oftener surfeited than emaciated; the face may be flushed; asphyxia of the extremities may occur; the muscles in general betoken profound tonelessness. In many cases the eyes retain what seem to be voluntary movements, and may searchingly wander round the room—a phenomenon which suggests considerable survival of sensation. Touch stimuli and stimulation of the special senses will usually elicit no response, but the application of mild pain is likely to elicit signs of distress. The reflexes in stupor are variable.

The condition is apt to be mistaken by friends and others for obstinacy, or what they vaguely call hysteria, and we may here note some points in diagnosis. Stupor, for example, must not be confounded with stupidity such as occurs in myxœdema, in adenoid affections, or in other cerebral "toxications," nor with semi-comatose states induced by narcotics. The history of the case must settle the diagnosis, with special reference to rapidity of onset in anergic stupor, the existence of predisposing causes of insanity, and of such immediate causes as explain cerebral neurasthenia — exhausting illness, shock, masturbation, etc.; but there can be no mistake if the physician attends sufficiently to muscular, vaso-motor, and trophic symptoms, all of which evidence much more profound depression of function than in mere stupidity. States induced by hypnotism have a much greater resemblance to stupor, and may only be discriminated from it by circumstantial facts. In catalepsy (*q.v.*) and in hysterical anergia, on the other hand, the symptoms of low-level depression may closely resemble stupor, but the mental impairment is slighter and transient. Coma, such as may occur in traumatism, apoplexy, the post-epileptic state, etc., may also be confused with stupor unless the history of each case be studied. Then there is the interesting condition described as lethargy, which is not a real stupor, but a condition of prolonged sleep. Clarke (*Amer. Jour. of Insan.*, Oct. 1891) describes a woman who slept for seven years, then had a remission of symptoms for seven years, then fell asleep again for a spell of thirteen years. In her lethargic phase, when not asleep, she seemed quite alive to what happened, did not soil the bed, ate food slowly, but did not speak. After these momentary awakenings she just turned round and went to sleep again. The fact that she did not speak for eleven years brings the case near to stupor. She always slept deeper at night.

It is interesting to observe that stupor is the mental disease in which a mistaken diagnosis is most likely; and the reason is to be found in its primary and essential symptom, irresponsiveness. The physician should consider this fact, that stupor is *par excellence* a primary affection, characterised by its negative symptoms, as illustrating the fundamental distinction between primary (negative) and secondary (positive) signs of mental disease.

Melancholic Stupor.—This affection is much commoner than simple anergic stupor. In the latter the onset may be rapid, may indeed, after a great shock, for example, be almost immediate. Melancholic stupor is a slower development as a rule, is attended by obvious mental pain from the first, and usually follows bodily illness, or some other neurasthenic process, such as overwork, exposure to extreme heat, masturbation, or acute insanity. Concomitant with the bad spirits there is the anergia—the inability to intend and to carry out intentions. Listlessness scarcely describes it. It is a disability as well as a disinclination for outward activities. In every case the impairment spreads outwards and downwards, and becomes evident in the flaccid muscles, torpid secretions, asphyxiated limbs, soft pulse, shallow respiration, trophic depression. These develop slowly, and as they progress, mental pain increases. In such cases suicidal feelings and attempts are common, not usually of a deliberately determined kind, but of the impulsive variety. Melancholic stupor is prone to fulminating (explosive) activities. These may take almost any form—a shout, a somersault, a blow, destructive violence, homicide, suicide, or an epileptoid fit. Melancholic stupors must always be carefully watched. The mental content, so far as we know, is largely one of passivity. Many patients recall, on their recovery, things that happened during their stupor, incidents which occasioned bodily pain, —a fact which, as was said, would indicate less anæsthesia than many authorities assume. Such cases end variously—often in recovery, which is frequently preceded by a maniacal phase, a low-level ideo-motor activity, which ushers in the recovery of the projective activities of the higher cortex.

Delusional Stupor.—As Clouston argues, stupor is very rarely determined by a patient's delusion. In a few cases, perhaps, a sudden and abiding obsession (fixed idea) may explain the patient's immobility, such as that he is dead, or that the least movement may bring about some catastrophe. In the vast majority of cases, however, the delusion is the patient's apology for his stupor and not the real explanation of it. Finding himself muscle-bound he invents an account of his state—that he is made of glass, for example, or that his veins are full of mercury, that his head has dropped off, that his body belongs to some one else. Crudity is characteristic of stuporose delusions. They amply bear out what I have said, that delusions are due to too little, and not to too much think-

ing. Stuporose cases do not make elaborate and systematised conceptions as a rule, but become possessed of simple, fixed beliefs, as immediate and unrelated as obsessions. In the sphere of muscles the characteristic mode is that of increased resistance; and a really good case of resistive stupor is a remarkable phenomenon. Passing from the anergic state of mere flaccidity the muscles become the site of distorted activities, and, assuming attitudes appropriate to what the patient feels and believes, will not be moved from them by anything short of dangerous force. I am not aware that any one has measured the tension in such cases, but to the casual observer it would seem appropriate to describe it, not in terms of foot-pounds, but in those of horse-power. As a rule restiveness, even in stupor, is aggravated by the mere presence of other people. It differs from that of melancholia in that it is resistiveness to an interference with a stereotyped attitude. The patient perhaps sits with his hands on his knees—an attitude of delusional safety, and retains that attitude in resistance to interference even to the point of fracture; or, if allowed, will wear the skin away by pressure. A melancholic resistiveness is much more general. If you tried to change such an attitude in a melancholic, he would probably lift his hands to strike, or his feet to kick, or would clutch at things near, or would run away.

Cataleptic Stupor.—Many stupors develop cataleptoid symptoms. I need here only point out the relationships of this variety. Catalepsy in stupor is, for example, the obverse of resistance. It is much nearer a direct development of simple anergia. In the stuporose muscles there is plenty of energy, but it is lying fallow for want of centrifugal stimuli. Unable to energise himself, the patient may, instead of becoming resistive, take on an exaggerated muscular facility, and the cataleptoid state is the gymnastic of that mode of attention. In vol. ii. of this work, p. 59, reference is made to the cataleptic muscular state. In stupor we do not have the completely waxen condition so often as simply a disposition to retain induced poses. The feel of the patient's muscles gives us more of the impression that she is voluntarily retaining the attitude which we have imposed upon her; but occasionally the *flexibilitas cerea* is present in some degree. In any case the plastic muscular state is not compatible with delusions such as have been indicated, but is usually associated, so far as we can gather, with mental torpor and vacuity. When it is obvious, asphyxia of the limbs and trophic impairments are not marked. Sudden movements may occur alternately with it.

Mania.—In stupor we have a disease which, as has been said, is to be described almost entirely in negative terms. Secondary and positive symptoms—delusion, grief, muscular resistiveness, catalepsy—do occur; but the primary features are the muscular and mental immobility. In melancholia the negative condition, the absence of outward activities, is evident enough, but we designate the disease by its predominant secondary symptoms—the painful self-regard. And in mania, also, it is the secondary symptoms, the excessive low-level activities, which are most obvious. Mania is in contrast to stupor—a diarrhœa instead of a constipation of ideo-motor activities in a brain which has lost its highest and controlling functions. For the primary condition of mania is, as in melancholia, an abeyance of normal outward relations. True, the maniacal stage is characterised by a diminished subject-consciousness and by an exaggerated object-consciousness. But the object-consciousness is on a low level and quite abnormal in its mode. The essential fact, then, is the abeyance of normal personality. That has to be repeated *ad nauseam* until physicians learn to have less regard to its secondary consequences; and it may be serviceable to illustrate it in connection with mania. For that purpose let us assume the patient to be one of an average mind, whose personality is concerned with the home and the earning of a livelihood as central elements in his consciousness. What happens in mania, then, is that he loses these attachments. His occupations become stale, he neglects his work, he disregards his wife and children, he destroys the peace of his home. If the attack be sudden, we do not think of it in this light, but in that case it is still more obvious that the patient has broken loose from his calling and from his home; he has dropped his career. And this failure of normal personality implies mental pain, and melancholia becomes manifest if, so to speak, the disease gives it time to develop. In many cases, however, the malady develops so rapidly that the melancholic stage is only an affair of an hour or two, and in cases of immediate mania the pain-stage seems to be as short as in a case of sudden death. In the great majority, however, careful inquiry reveals that, for some time past, the patient's spirits have been at least fitful—the sign of incipient failure in the mechanisms to which I have referred.

Passing slowly or rapidly through the stage of pain and subjective over-attention, the patient soon develops an excessive activity in ideo-motor functions on a low level. In melancholia there is in general a depression of cortical function, both high and low. In mania there is activity to excess on levels lower than the normal highest. And that bespeaks a pleasurable tone of spirits which has, moreover, its characteristic gymnastic. When the maniac is at his quietest his muscles assume the extensive mode. The wide-open eyes, the expanded mouth, the head thrown back, the shoulders open, the spread fingers, the erect carriage, the straight knees, are in strong

contrast with the gymnastic of melancholia. And the exultation is intelligible; for the dynamical conditions of pleasure are fulfilled—rest of the devitalised mechanisms and full activity of the energetic—exercise of the imagination, of the special senses, and of the voluntary muscles. And even in mania the systematic nature of the disorder can generally be demonstrated. It is impossible here to expound the statement, but mania is not intrinsically incoherent. The maniac's sentences seem incoherent to us, but they are coherent to his surviving mode of consciousness. Could we obtain a complete bird's-eye view of all that happens in his cortex, we should discover that his disordered activities, even when peripherally initiated, and still more when they are centrally determined, follow along paths which are organically associated. And the big types of mania are obviously systematic developments. The following varieties are those described by Clouston. But the disease is so gross, so obvious, so fully treated of in the text-books, so impossible of private care, that we need only point out salient features and remark a few of the relationships of the affection.

Simple Mania is often not diagnosed in its early stage. There is a general exaltation of cortical function. The patient is restless, active, fanciful, pleased with himself, but often quarrelsome. This stage is characterised by great activity of the high-level functions short of corrective judgment and coherent purpose. The consciousness teems with extravagant ideas, hopes and schemes, and revels in excess of affections. Now and onwards we must note the unrestrained activity of the objective relations. Every little change in the environment suggests something to the patient's restless mind. A clever companion, who cares to lead the patient on, will elicit an apparently endless flow of conversation, quite coherent, but grossly extravagant, and may lead him into any madcap adventure—a visit to the crowned heads, or to the North Pole, the purchase of an army of elephants, or of half the coal-fields of Europe. But let the guidance of the companion be withdrawn even for a moment, and the exuberant interest of the patient will fall a prey to the next suggestion his environment offers. Facility and fickle enthusiasm are the features of this stage.

Acute Mania.—In acute mania the loss of function becomes more obvious. The patient is incapable of ideas and of purposive activities, even of the insane kind, which occurs in simple mania. Consciousness now is apparently on the Rolandic level. At least the patient's activities are almost entirely sensori-motor. She—for women offer the most exuberant symptoms of acute mania—has passed gradually from active ideas and projects, and the attention is occupied wholly with sense-perceptions or with simple movement, or with both. Over-attention to sights and sounds and smells—the sensory mode of acute mania—is in my opinion less desirable and apt to become a chronic insanity with hallucinations. As a rule, however, objects perceived excite an immediate motor response. The patient sings in answer to the birds, knocks over vases, shouts the name of each thing she sees, blows her nose fiercely at a smell, poses, jumps, and gestures appropriately to the stimuli of which she is conscious. And if this sensori-motor relation obtains, such a case is more easily led back, under appropriate treatment, to orderly and purposeful activities. Other cases, again, are of a predominantly motor type, whose muscular activities seem to be subjectively determined. The outward relation is at its acme in such cases, at least quantitatively. The patient dances and gestures like a dervish to the point of exhaustion, or talks or shouts or sings incessantly. In the latter case ideation is at a minimum, and words, not sentences, pour from her lips, though she may repeat set phrases or snatches of poetry or song. Quite often she will suffer a *diarrhœa verborum*, whose only coherence is a rhythmical or rhyming or alliterative or other non-ideational association. These are the cases most likely to become exhausted, and liable to end in stupor or in acute delirious mania or in dementia.

Acute Delirious Mania.—*Bell's Mania.*—In acute delirious mania the reduction of cortical function is still greater, and is accompanied by grave bodily symptoms. The mental condition is one of delirium. The patient is unconscious of things around her as such. She stares at them, feels them, picks them, as in a dream, moves restlessly about in a wandering manner, as if looking for something, or as if trying to get away even through walls and doors, and only mutters if she speaks at all, or cries feebly or whines. And meanwhile she is physically exhausted and febrile, appetite is gone, the tongue is dry and heavily furred, there are sordes about the mouth, the breath is foul with an ante-mortem odour, the pulse fast and irregular, the respiration shallow, and not proportionately rapid. Sleep is impossible. The case is now one for most careful nursing and sustaining food, which usually must be administered by the tube; and, in my opinion, rest should be obtained by mechanical restraint. This condition sometimes develops rapidly, or, less commonly, follows a prolonged acute attack. The symptoms certainly suggest, over and above exhaustion, some cerebral toxication such as we suppose to be the part cause of other febrile insanities. No one knows whether the toxins are the products of extreme nerve waste or of paradigestion, or from some other source.

Delusional Mania. — This condition, as Clouston observes, is to be distinguished from delusional melancholia and from chronic delusional insanity, only by the fact that the

patient's conduct is maniacal. Indeed there is no line of demarcation, for the delusions in these cases are often of a distressing character. Delusions abound in simple mania, but they are fleeting. Abiding delusions now referred to are the conceptions of a mind in chronic mania, such as express resentment for supposed injustice—that people have designs upon the patient's property or character, that certain people in their jealousy have conspired against him, that, while he is detained in the asylum, his enemies are wrecking his home and destroying his wife and children; and excitement and violence supervene.

Chronic Mania.—Chronic mania is also an arbitrarily determined variety. It is characterised by continued persistence of subacute excitement. Some patients remain violently active on the sensori-motor level for several months; but not many constitutions can stand it. As a rule the acuteness of the activity wears off and ideation is, to a certain extent, restored. The patients then go about prone to restless and extravagant conduct, and to active and fleeting ideas. In this condition we have, *par excellence*, the insane survival of the constructive mode of consciousness. Usually demented in some degree, the chronic maniac may spend his days in fashioning curious idols which embody for him important attributes, in inventing and constructing hopelessly unnecessary appliances, or in designing and laboriously completing some handiwork which he is sure you will be glad to retain as a souvenir of his regard for you. The tone of feeling may be happy or it may be resentful—the hilarious and the angry types of acute mania as distinguished by G. M. Robertson. The imminence of dementia is usually obvious in such cases.

Ephemeral Mania.—*Mania Transitoria.*—This variety is distinguished by its sudden onset and offset. It must be regarded as an epileptic equivalent. The patient suddenly suffers an abeyance of the functions of normal personality, but does not fall to the ground or lose consciousness in the ordinary sense. Instead of motor convulsions—the typical clonic spasms—the explosive activity is manifest in sensori-motor violence often destructive, such as occurs in ordinary acute mania. The duration of the attack is to be measured in hours. Then, after something like a post-convulsive coma, or sleep, the patient awakes to resume his normal activities.

Recurrent Mania.—To the above varieties we may add that of recurrent mania—states of subacute excitement associated with a delusional point of view, which occur periodically both in men and in women, but especially in women, and generally related to menstrual functions. These are usually chronic cases, manifesting between times a fairly sane consciousness but often slightly demented, yet subject at the menstrual period to recurrences of acute excitement. Such periodicity, common in nearly all forms of insanity, betokens a preponderance of inherited proclivity. Nothing further is to be said about these cases except as regards prognosis. Only when the patient has survived at least two periods without a maniacal relapse, may we assume that her convalescence is assured.

Alternating Insanity.—All intelligent persons alternate to some extent between phases of happiness and of unhappiness. These are modes of consciousness which respectively regard the environment as more or less at the disposal of oneself; or oneself as more or less at the mercy of the environment. Cheerfulness persists just so long as we stand up to life; apprehension darkens into sadness in the minds of those who go down on their knees before it. And these two attitudes of mind severally distinguish melancholia and mania. In melancholia the mind is reduced to a primitive level of fugitive consciousness; in mania to one of foolish activities. But in many cases—and these are always interesting—the insanity reveals a systematic and more or less regularly periodic alternation between the depressed and the exalted phase. Such cases usually, indeed nearly always, have a spell of sanity in their cycle, and that is a strange and interesting fact. The patient recovers from melancholia perhaps and seems quite well for a period; then, with a scarcely perceptible stage of depression, passes into mania for a period; then to melancholia again, and so on. And nothing so impresses upon one's mind the idea of the systems of subconsciousness as one of these cases. Here is a man beneath the surface of whose mind is an organised system of fear, beset with gloomy feelings and with a varied assortment of terrors. Each time that he becomes reduced to this fugitive level, he feels the same kind of grief, entertains the same kind of delusions, and wears the same expressions and attitudes as before with very little variation. But he has also a subconscious phase which has reference to his surroundings from the point of view apparently that the world and all its delights are entirely at his disposal—a phase of consciousness that revels in primitive and puerile activities. And again, each time he reverts to this maniacal phase, he behaves very much the same as when last he was reduced to mania—makes love in the same silly phrases, entertains the same ridiculous projects, adorns himself similarly, walks with the same jaunty gait. And as we note these phases of consciousness, their distinctive gymnastic, and their coincident and characteristic trophic reactions, we cannot but assume that, in such a patient, subconscious mechanisms are deeply organised to represent them. Moreover, the melancholia of such an one does not essentially differ from that of a patient whose

disease never proceeds to mania; nor does his mania materially differ from that which never alternates with melancholia. We are forced to the conclusion then that, in the minds of all who become insane, there was a systematised melancholic or a systematised maniacal character in their subconsciousness or both. But we are probably safe in assuming that these subconscious arrangements characterise the brain of the human species, and that they would inevitably be revealed in any one who, having lost his acquired personality by disease, was reduced to a level of consciousness of an emotional form. (The same kind of things might be said of delusional types.)

Over and above the impression which alternating cases give us of the systematic nature of cerebral arrangements on levels lower than that of sane personality, we must be struck by their periodicity. To watch a man through a *folie circulaire* is to have it suggested that the human brain works in cycles, that each cycle reveals several phases of consciousness in strong relief, almost as dissociated as in the insanity of alternating personality; and that cyclism must be briefly considered before we describe the types of the disease.

Periodicity.—Nearly all brains that are active, and all that are constitutionally disordered, manifest a more or less regular cyclism. Nor is such periodicity a new or a strange thing. It pervades the whole of organic life. For the primary factor in its occurrence we must go back to astronomy. The same rhythm by which the stars in their courses are set, and which brings our earth's surface near the sun at definite intervals, determines also the orbit of man's brains—sets it to a measure that rises and falls with a regularity that we scarcely notice except when disorder reveals it in excess. These immense, unconscious influences are quite beyond our reckoning, but we may surmise that our terrestrial nervous systems would be grievously upset if they were suddenly transported into the rhythm which besets Mars or Venus, or still more that of Jupiter, as surely as many suffer agonies of discomfort on quitting the stillness of land to embark on a swelling sea. And though the ship's mechanical motion has a very short wave-length compared with day and night, and these again as compared with summer and winter, yet are the latter further reaching in their effects. We must think of the diurnal variations of our atmosphere as a great pulse-wave of heat and of light which determines all the nutrition of our earth's surface, and which imposes a rhythm upon all organic activity, from the production of chlorophyll to the weaving of dreams. All over the world the heart muscle and the muscles of respiration are contracting at a measure which, taken in relation to the weight of the individual, bears a constant ratio to the time of the earth's revolutions; and, over and above these regular ripples of activity, our massive movements also occur in diurnal wave-lengths. The next big rhythm is of a monthly type, both in men and in women. The lunar coincidence is the only factor which we can suggest as an explanation of that. At all events the fact is that, in nearly all women and in most men, cerebral changes occur in monthly periods. That rhythm, and the largest of all, the seasonal cycle, has an indirect influence on cerebration reflexly through the sexual and the reproductive functions. Under the influence of natural selection, organic changes in the female have become stereotyped in a rhythm which determines a rise of sex-consciousness in the spring, or, by an elaborate adjustment to the period of gestation, makes the birth of the offspring likely to correspond with the most suitable climatic conditions for its early life. From reproductive habits of a seasonal type the human race has of course departed. But the constitutional changes which are so evident in lower animals as of a seasonal nature, are the inheritance of our species also, and are organised throughout our whole nature. For the teleology of female changes applies also to the male. Animal life, in males, is largely a reaction to female states, and the organic energies of the two sexes usually wax and wane in harmony.

From the strict type, the human race is obviously diverging as civilisation diminishes the direct influence of climate upon health. A woman's concealment of menstrual symptoms is but a crude sample of a general levelling process which polite society enjoins upon men and women. Even pregnancy and lactation are considered impolite, and, with the general suppression of these and other appearances of reproduction, periodic reactions in the race are being obliterated. Equableness is now indeed a feature of cerebral stability. But in incidental insanities and in abnormal mental reactions periodicity is conspicuous. Clouston and others regard these periodic exacerbations as an almost sure sign of a neuropathic diathesis. In other words, while all constitutions are subconsciously of a rhythmical organic habit, only weak brains fail to rise above and to conceal these alternative exaltations and depressions.

Periodic Mania.—The recurrent variety of mania is the first form of periodic insanity, though melancholia may also manifest regular exacerbations. As was indicated, *Recurrent Mania* is most obvious in women, because of its relation to menstruation. But it occurs also in men. Sometimes it appears that the sane period signifies an access of energy and that the mania occurs on the subsidence of vitality. More often the facts of the case suggest that sanity suffers a periodic interruption occasioned by an access of vitality in low-level mechanisms. I have at present two cases which certainly offer a contrast. One of them, neurasthenic

and anæmic, suffers a periodic relapse into mania. The other, robust and buxom, seems a chronic maniac, interrupted by periodic relapses into ephemeral sanity.

Circular Insanity.—The classical variety of cyclical insanity, however, is one which alternates between melancholia, mania, and sanity. These are cases of gross insanity specially interesting to the general practitioner because, if the rhythm has a grand wave-length, the patient may return home for months or even years, and it then becomes the practitioner's duty to watch for the relapse from sanity. And, while these cases are, on the whole, of a bad prognosis, a great deal may be done to postpone the relapse.

Theoretically, these cyclical cases might be expected to manifest a periodic reduction of personality, with melancholia as a half-way stage between sanity and mania both on the down-grade and on the upward return, thus: sanity, melancholia, mania, melancholia, sanity. But the disease is very irregular and the melancholia is often not observed, especially on the way from sanity to mania. Moreover, the periods of duration of each phase are, as a rule, irregular. The rhythm may be set to a measure of anything from a few hours to a few years. As types, however, we may discriminate three: those presumably determined by diurnal variations of vitality; those whose rhythm approximates to the monthly type; and those which are some modification of seasonal periodicity. Clouston in his text-book has a classical description of the last form. The periods were as follows:—

I. Cycle.—Mania, 6 mos.; melancholia, 3 mos.; sanity, 6 mos. = 15 mos.
II. Cycle.—Mania, 12 mos.; melancholia, 6 mos.; sanity, 6 mos. = 24 mos.
III. Cycle.—Mania, 10 mos.; melancholia, 6 mos.; sanity, 8 mos. = 24 mos.
IV. Cycle.—Mania, 13 mos.; melancholia, 6 mos.; sanity, 14 mos. = 33 mos.
V. Cycle.—Mania, 24 mos.; melancholia, 12 mos.; sanity, 15 mos. = 51 mos.
VI. Cycle.—Mania, 3 years; melancholia, 2 years; sanity, 1 year = 72 mos.

After that the patient suffered an almost fatal neuralgia, followed by some years of sanity. In all such cases it is not so likely that a particular phase will have a duration equivalent to a given seasonal or menstrual period as that the change from one phase to another will occur about the zenith or about the zero of vitality. From these grand (seasonal) variations down to the menstrual types, intermediate varieties abound. So also between these and the petit (diurnal) forms. Clouston quotes another case, "D. D." æt. 49, "who for twenty-six years was subject to regularly recurring brain-exaltation every four weeks, almost to a day." In others, the cyclism is of a fortnightly or weekly wave-length or thereabouts; and, in still others, diurnal or shorter. Many patients go to sleep in melancholia and awake to mania or to sanity, which again will be only of an ephemeral duration. Others suffer a forenoon melancholia and an afternoon mania. And in all such phases we have to note motor, sensory, delusional, and volitional concomitants. In some, stupor characterises the melancholic phase, and in others, delusion; so also the mania may be simple or acute, delusional, querulous, or hilarious.

Over and above the care of these alternating cases in their sane phases, the physician may render great service in detecting cyclism in neurotic youths, and in prescribing a mode of life which will modify it.

It is interesting, as was said, that most of these cases come back to sanity at some arc on their cycle. It is comparatively rare for a patient to alternate periodically between melancholia and mania. Also, why do we not often find alternating cases with fixed delusions characteristic of each phase? In alternating personality (double consciousness) we have such a condition, though it is not markedly periodic. It is considered later. (It will be noted that periodic or recurrent mania and alternative personality may be variously classed. Insanities are not capable of exact and exclusive classification.)

Delusional Insanity.—There is no kind of insanity about which so much confusion prevails at this moment and so much difference of expert opinion as about the varieties now to be considered. The practitioner who tries to keep himself in touch with the literature of insanity must be in a condition of great perplexity concerning delusion, unless he is shrewd enough to appreciate the fallacies which beset asylum work.

To unravel this tangled skein will be the work of many years of careful inquiry and of clinical and pathological comparison. The following account of the subject must therefore be regarded as tentative, and it is right to say that many advanced students of delusion would regard it as quite misleading. Some fallacies, however, are so important that they must be pointed out. In the first place, most authorities describe delusional insanities chiefly from observation of asylum cases of long standing. These, it need hardly be said, are of the least practical importance, though of profound interest from a scientific point of view; and, compared with delusional states of a slighter degree, such as are met with in general practice, they are few in number. Another source of much error is that we attach far too great an importance to the refinements of foreign observers. Magnan's insanity (progressive systematised delusion) very rarely occurs in this country as a typical form. Again, we must note that very great confusion has arisen from the perverted use of two terms

—paranoia and degeneracy. Any one who cares to make reference to recent English work, to say nothing of American and continental, will wonder why we have not entirely discarded the term paranoia. One could hardly conceive a greater discrepancy in scientific opinion than occurs for example in the accounts of paranoia given by Clouston (last edition), Bevan Lewis (do.), Campbell Clark, and Macpherson, in their text-books, and by Conolly Norman in Allbutt's *System of Medicine*. Paranoia, however, is the subject of a special article by Macpherson (*q.v.*), and it is best that we should restrict the term in this work to the interpretation which he puts upon it. And as to degeneracy the same confusion prevails. The degenerate is, in fact, as has been said, very elusive except in his superlative degrees, and we err on the safe side if we withhold our diagnosis of him except in that form, that is, as a criminal, either in intention or in fact, a person incapable of morality and usually of a correspondingly degraded physique.

Considering that the human mind is admittedly and properly regarded as having three great aspects, thought, feeling, and will, it would be strange indeed if a great many cases of incidental insanity were not chiefly characterised by disordered thinking, without a proportionate volitional and emotional disturbance. All the same, the prime fact about intelligence is its inevitable correlation with all other aspects of mind. No one proposes, or could possibly expect, that a patient should reveal simple delusion. Bevan Lewis goes so far as to say that "monomania is evolved out of melancholia and maniacal perversions—as a special derivative of these conditions, and as one of the terminations in chronic insanity." So far as I know, he is alone in that opinion. But, in any case, monomania is descriptive of but a few delusional cases. Before a patient becomes delusional, that is, when his thinking mechanisms fail in their higher phases and become active on a lower level, some mental pain, of however short a duration, must be presumed, as it always must be in states of cerebral exhaustion. But that does not constitute melancholia. Nor do delusional cases always conform to what we have agreed upon as the secondary characteristics of mania—an excessive activity of an outward form on reduced levels. Delusion is not such an excessive activity. We cannot repeat too often that delusional cases as a rule suffer from too little, and not from too much thinking. Patients who entertain delusions, if they thought enough, would realise that they were believing nonsense. There are many cases of melancholic delusion, or delusional melancholia, just as there are others of maniacal delusion, or delusional mania, cases in which, with the essential signs of melancholia or of mania, disordered thinking is conspicuous. So also with stupor. Such cases have been referred to. But those are not the delusional insanities now to be considered. They constitute the connecting links between melancholia, stupor, and mania on the one side and simple delusion on the other. For all the types of insanity that we are considering have alloys which closely connect them one with another. Even in one case we may observe insanity manifest in melancholia, stupor, mania, and quiet delusion in successive stages.

When a patient "loses his head," that is, when the mechanisms of normal personality fail, he may be reduced to a level of painful self-regard, which is melancholia; or suffer also an abnormal resistance in the mechanisms of expression (projective), which is stupor; or become outwardly active on a lower level, which is mania; or he may suffer a rapid dissolution of all his mental functions, which is dementia. But another alternative remains of a systematic kind, which may characterise his insanity. He may be reduced to a level of consciousness in which his attention is chiefly and conspicuously, and to an excessive degree, occupied with beliefs. That is the form of insanity—a disorder of opinion—with which we have now to deal. The primary form of such insanities is a condition of confusion, in which the patient does not know what to believe. The secondary form is delusion, in which he believes the wrong thing. Both in confusion and in delusion the mental content may be one of depression, or, with grandiose delusion, it may be one of exaltation, but in neither case need there be that degree of depressed activity or of excessive activity which characterises melancholia and stupor or mania.

Confusional Insanity.—On this subject the unwary physician is apt to be misled by the contradictory and uncritical accounts of confusion which prevail. In particular, many observers conclude that, because a patient's statements are confusing or confounding, he is therefore confused. But the reverse is usually the case. A patient may believe several irreconcilable and even contradictory things and yet be quite sure about each. That is not a confusional state. The term *Confusional Insanity* only applies to patients who are bewildered and in doubt and hesitating. That is a state of mind which occurs as a passing phase in nearly all insanities. But it sometimes persists for a long time as simple confusion, and may resolve, so that the patient then recovers; or it may go on to delusion generally with hallucinations. The disease is analogous to *folie de doute* or swithering insanity—a condition of bewilderment and hesitation in conduct.

Confusional patients always suffer from an excess of suggestion. They cannot entertain simple beliefs, but are beset by innuendoes and corollaries. In all such cases there are cerebral

activities of a form very near hallucinations and obsessions and illusions. Illusions are misinterpreted sensations; hallucinations are sensations or perceptions automatically occasioned, that is, without an apparent objective stimulus; obsession is an idea which occurs again and again, and obtrudes itself irresistibly upon the attention; delusions are mistaken beliefs, whether referring to perceptions or to oneself, or to things in general. But in insane processes there are no hard and fast lines between these various morbid activities, and the merging of one into another is clearly apparent in confusional cases. Ideational activity of an unrelated form—the occurrence of words and meanings in the mind without a proper content—are due to morbid processes in mental areas exactly comparable to hallucinations such as voices, bells, singing, and visions, which presumably arise near the Rolandic areas or below them. A gentleman who had been working hard in a hot climate told me lately that, on several occasions when resting, words, not in any recognisable voice, and having no apparent coherence, crept upon his attention in a string, such as "Mine—hut—sweet—contract—lie—," like a telegram in cypher of which he did not possess the code. The moment he turned his attention full on the process the representation ceased. If he had attributed these words to some voice, we would call them hallucinations of hearing; if he accepted them as significant and believed nonsense because of them, he would be suffering delusion. And in confusional insanity, without hearing actual words, meanings, ideas, and suggestions occur to the patient's mind and have to be explained. In particular, confusional cases suffer from a morbid process very analogous to illusion in that they lay an emphasis on certain phrases that they hear that was never intended, and ponder over them with care and perplexity. Some confusional cases are insanely mystical. The patient begins to doubt self-evident facts, cannot accept the material limitations imposed upon our senses, and constantly seeks a mystical and subtle explanation of the most simple things. These metaphysical disorders usually have a religious reference. Such patients are often slow to accept a dogmatic delusion, they simply regard everything as an open question; but such a state of mind is quite unfit for society, and it is on the way to gross delusion if not corrected. A patient, for example, who wonders whether some one else can occupy his mind, and who occasionally supposes that the things he is saying and doing may not be his own, is very near to delusion. Many confused patients are haunted by that suggestion of an indwelling spirit. I have voluminous notes written by one of Dr. Clouston's patients of this class—essays upon such problems as that of the "existence of the real objective world, which exists, I suppose, objectively and subjectively, to God and in God." His mind was of extraordinary quickness and subtlety, his nature very gentle and generous and affectionate. I often think of him as one whose mental disease is to be explained on the hypothesis suggested in a previous paragraph—that the organs of his personality were a good deal too much in advance of the standards of our time. He suffered from an alternating disorder of the intelligence. We called him well when we had induced him not to think, but to engage his muscles. When he took to thinking he usually became painfully confused and bewildered. Sometimes he then went back to a muscular life, in which he found relief; at other times he gave himself over to some expansive truth such as that "God is love," that "all thought is one," that "persons are inseparate," etc., and, revelling in the developments of such an idea, he suffered what was really a mild ecstatic mania for a few days. In that phase he could hardly be said to think. Everything was clear to him; he knew all that he cared to know; he did not require to consider. Most confusional cases, however, are perplexed by much less subtle imaginings and occupied by much more mundane suggestions. Two cases under my care, one a man, the other a woman, were very far from occultism. They were perplexed usually about quite trivial matters—wondered if somebody meant anything personal when he said that the day was overcast; or if that remark at table, "Make yourself at home," could signify a good deal more than appeared; and so on. A great many patients from overstrain or shock go no further than simple bewilderment. They complain that they cannot understand things as formerly, that in reading or conversation some irrelevant suggestion crops up and confuses them, and that their memory is precarious. If such patients can be taken in time, compelled to rest, and not allowed to think, recovery often follows quickly. As soon as the patient begins to ponder over these cross-suggestions he is in risk of becoming delusional, or if, without entertaining these fleeting ideas, he allows himself to mope over the facts of his case, he is apt to become melancholic. The primary lesion in this as in swithering insanity seems to be a destruction of common sense. That characterises all delusional states.

Systematic Delusions not Progressive.—Patients who entertain absurd suggestions for any length of time usually end in gross delusion. Their mistaken beliefs are systematic in the sense that they are elaborate conceptions, supported by a mass of supposed evidence, and they are systematically expressed in conversation and in conduct. But in many cases, though perhaps not in most—which is doubtful—they are not progressive. That is, ill-health and cerebral neurasthenia, characterised by doubt and by delusion, may be recovered from as

other insanities are. Under appropriate treatment the delusion obviously loses hold upon the patient's mind; prevented from expressing it, he lets it wait; induced to occupy himself with other things, he loses interest in it; for some time, perhaps, he still thinks it may have been true, then finally he regards it as folly. These episodic delusions are much more important than progressive delusions, because of their frequency and because of their curability. The topics of them are innumerable, and we can only glance at the more common. Nor need we pause to remark the coincident effect upon the spirits which such delusions have—sometimes depressing, sometimes exalting—nor their effect upon conduct, for these can be inferred with approximate accuracy.

Delusions of bodily illness are very common, and the patient, though not the subject of melancholia, entertains the kind of beliefs which have been indicated as common in the visceral form of that disease.

Delusions of seduction often occur in women at or about the climacteric, and in girls at or about puberty. Such cases may assume a medico-legal importance. Short of these, many aggrieved spinsters come to believe that they are the victims of some one who has wrecked their affections and broken a promise of marriage. There is generally some circumstantial foundation for such delusions. So also with *delusions of pregnancy*, which are most apt to occur in unmarried women who have cause to be afraid that they are with child, and in married persons who are afraid that they are not. Of a similar kind are the delusions with which we are familiar in hysteria and which refer to a supposed *loss of power*. The patient believes, perhaps, that she cannot walk or cannot hear properly, that memory is failing, that she is entering on her dotage or becoming insane. These mistaken beliefs may occur independently of a hysterical condition or persist beyond it. All such delusions referring to bodily states are occasioned in part by peripheral or systematic stimuli, but the misinterpretation of them is determined by central disorders.

Religious delusions are of another class. The patient believes, perhaps, that he has a special mission conferred upon him by the Almighty by immediate revelation, or that a universal crisis is at hand, or that God is personally displeased with some one of his paltry misdemeanours, and threatens some disproportionately dire revenge. Others may be described as *social delusions* in belief of which the patient supposes, for example, that he is on the verge of making a great fortune or of losing it, or that it transpires that he is after all of noble birth, or that he is an outcast, and that society is sending him to Coventry. Then there is that strange condition to which both men and women are liable who believe or pretend to believe that they have been attacked by robbers or burglars, or fired at, or poisoned. All these general delusions are to be attributed to central rather than to peripheral disorders.

Monomania (*Fixed and Progressive Delusions*).—Dr. Macpherson describes paranoia in a special article (*q.v.*) and includes two clinical types. One of these is the systematised progressive delusion (Magnan), characterised by absence of inherited or previous insanity, occurring, that is, in strong-minded persons with a stage of inception, one of persecution with hallucinations, one of ambition or grandeur, and one of dementia. The other is the paranoia of the degenerate—a form of delusional insanity which may simulate Magnan's type, but which lacks its constancy of feature and its tenacity.

Some would have it that no more need be said about fixed and progressive delusions, but a host of accurate observers in this country recognise that the above two classes still exclude a great number (perhaps the majority) of fixed British delusions. The distinctive feature of the above two types, it will be noted, are, in the former, absence of recognisable predisposition and remarkable constancy and tenacity in the progress of the delusion; and in the latter an obvious diathesis and much variability in the progress of the delusion. Exceptions in both respects abound. There are, on the one hand, many apparently strong-minded persons, not obviously of the insane diathesis, who become the victims of a systematic and progressive delusion which does not even approach to Magnan's type in its course. Some of these are delusions of exaltation not preceded by a stage of systematic persecution. Others are cases of delusions of persecution who die of a ripe old age, perhaps softened and not so angry, but still showing no signs of passing into an exalted stage, or who obviously miss it out and become demented before they die. And, on the other hand, there are still a larger number of exceptions—cases with well-marked predisposition to insanity, whose delusions are very systematic and very tenacious. These cases, as was said, are all unimportant except from a scientific point of view. You cannot do much in the way of treatment for the King of all the Continents, and the longer he reigns the less likely is he to abdicate. We shall therefore merely indicate the nature of these insanities, and in so doing shall use the old and much-abused term, monomania.

Monomania of Persecution.—A systematised and progressive delusion of persecution often arises after an attack of mania or of melancholia, or it may be occasioned by bodily disease or other cause of malnutrition, or it may develop gradually in a suspicious mind. Reverting to what has been hinted in considering Alternating Insanity and previously, we have to regard delusion of persecution as an expression of the fugitive

consciousness in the sphere of ideas, as melancholia expresses it emotionally. The patient gets to feel that he is at the mercy of his environment and that he is being worsted. Querulousness and grievance-hunting, as in Magnan's insanity, characterise the inception of the disease, and, by degrees, insane suspicion ripens into gross delusion and persists. From an early stage the disposition to believe evil is supported by peripheral processes, by paræsthesias, or by hallucinations. The exact expression which the patient finds for his sense of persecution matters very little. But we have to note two directions in one of which nearly every monomaniac of persecution seeks his explanation. Some patients believe themselves to be the victims of a conspiracy of known people, or of one or two people. Others refer their supposed evil flight to unknown and unknowable agencies — mesmerism, electricity, evil spirits, or drugs. When the patient insists upon the bodily evidences of his persecution we may be sure that he has some real trouble to complain of. Take this letter, for instance, quoted by Clouston—a good sample of such complaints : "Forced dreaming, forced vomiting from the stomach, forced glut vomiting from the throat, cold shivering by the forced thinking, sweating done in the same way, pains in the stomach any way they think. I think it is time that this way of punishment should be stopped. . . ." These "punishments" are obviously bodily stimuli. And the quotation, in the recurrence of the word "forced," illustrates a not uncommon symptom, the use by the patient of a kind of slang (neologism) which he invents or adapts to describe his sensations. I have a patient now whom it is impossible to understand until you have learnt his vocabulary. He believes himself persecuted by what he calls "Thugs." But they are not Thugs as we know them but evil spirits who haunt the air, and who can even come at him through the earth, the trees, or walls. "Doctor," he writes, "I am at my last point. Such work these people are getting in on me as you can't possibly know. They have taken the outer circle, and they are verging more and more, prising it for all it's worth and far more than is legal." So also in cases cited by Campbell Clark in discussing Insanity of Persecution — "That I have got attraction to my frame, medical attraction to the atmosphere; that they are taking me in from Glasgow, doing as I am and has done; that they have controlled me to mechanism, steam or wind, gauging of the atmosphere; that I have controlled my frame to his caturging or rifle pits in Lenzie" (from a letter by a male patient). A female patient uses this same word "frame." "They took in my frame in concealment with their weapons." "They cannot telephone without a female frame being taken in." "I didn't come to expound the weapons and Mr. Tennant will let out my frame." These neologisms are of interest as suggesting feelings and ideas which no known words are sufficient to describe. And when a patient has these quaint paræsthesias, or when hallucinations occur in explicit support of the delusion, recovery is not to be expected.

Monomania of Exaltation.—The incidence of fixed delusion of grandeur may be as in monomania of persecution. There is a striking difference in the progress of the disease which has not received sufficient attention. When patients conceive delusions of persecution they do not, as a rule, change their identity. A few cases become Cain or Ishmael, the Scarlet Woman or some other unfortunate. But in the great majority it is still, say, John Smith or Eliza Jones who is suffering persecution. In connection with delusions of grandeur on the other hand, patients almost invariably assume a changed identity. They may become a royal duke or Jesus Christ, the Empress of India or the Wife of God, as if to account for the good fortune which they are persuaded has befallen them. But they almost never believe in a delusional good fortune befalling John Smith or Eliza Jones. When a patient conceives a delusion of good he almost invariably changes his identity and assumes a new title: those who conceive delusions of evil, retain their own identity, but assume that other people have taken on a new character. For a difference so notable, the only explanation that offers itself is the very obvious one that it is a predisposition of all human nature to throw the blame of a misfortune upon one's neighbours and to attribute to oneself all that is of good.

Another point requiring explanation is as to the cause of a sense of well-being in disease. We can understand the patient who, in failing health, feels bad all over and takes a gloomy view of everything. But how does disease occasion a sense of good fortune? In phthisis, in general paralysis, in alcoholism, a delusive hopefulness is often to be noted, and a similar anomaly presents itself in simple mania and in delusion of grandeur. In the latter instance the patient is on the rapid way to dementia in all probability, and yet he feels grand. The explanation of all such feelings probably is that, in a brain so diseased, the physical conditions of pleasurable feeling prevail. These are, as has been said, a series of reactions in mechanisms which are not called upon to discharge more energy than they can afford. In simple mania, in general paralysis, and in delusion of grandeur, the exhausted mechanisms are at rest. The patient does not even stimulate them by self-examination. And the lower, unimpaired centres are active—especially those concerned with actions and those which subserve imagination. That would imply that the melancholic brain, and the brain which suffers delusion of

persecution, is less deeply disordered than the brain of mania or the brain which conceives delusions of grandeur. The latter have lost nearly all self-consciousness and are the seat of a low-level activity. They have dropped their exhausted personality. The clinical facts of these grandiose cases need not be elaborately reviewed. Filled with a sense of good fortune, the patient believes that he is a person of great rank or of great wealth. As always, his conduct does not really square with his delusion. In particular he does not demand with consistent dignity that other people should acquiesce in his belief. His mind, so reduced as to believe the preposterous about himself, is demented enough to be facile and easy-going. Nearly all his expression of his delusion is in the sphere of appearances—in dress and in manner. Believing himself to be heir to the crown, he may assume a pompous air and may don a royal dress. But he smiles if you treat him as a lunatic, his crown is probably of straw and his royal robe a tawdry thing in tatters, he never concerns himself with the serious affairs of state. And the same superficiality and insane insincerity characterise patients who believe, for example, that they are Christ, Shakespeare, Crœsus, or Sandow. All such cases, when the patient assumes a new character, the personification of some kind of grandeur, and abides by it for years, are likely to become demented or to die in their delusion. Such insanities signify a reduction to a level of consciousness which is the converse of the fugitive—a refined mode of the predatory state of mind; an ambitiousness which regards all that the world holds of good as rightfully its own.

Alternative Personality—(*Double Consciousness*).—Cases of alternating personality are endlessly interesting and suggestive. And their special attraction, like that of some other mental disorders, seems to be because they exhibit in exaggerated form modes of consciousness with which we are familiar in sane life. We find it useful to take the simple mind as our normal standard and select its prevailing interest, say the domestic, as determining personality. But the more active human minds are far too complex to admit of such description. Such persons especially as are aware of a conflict between what they call their better selves and other phases of themselves which they do not understand and cannot manage, can at least be said to have interests and pursuits which are without their personality. Indeed we must note a type of persons whose different phases seem hopelessly discrepant and are in fact not at all bound by any unifying process in their consciousness. Such people can be said to have two or more personalities—as difficult a metaphysical conception as that of the Trinity. And this dissociation of the various phases of a mental life, though it has not received much attention from alienists, is a very important factor in insanity, even when there is no obvious question of morals involved. The dislocation which such minds suffer is metaphorically described by Robert Louis Stevenson in his "Strange Case of Dr. Jekyll and Mr. Hyde" as a profound bodily disturbance. But even when there is no such conscious antagonism between the two or more personalities, there must be a cerebral dislocation, a dissociation of two or more great kinds of activity, and such brains, whose parts are not subordinate to a central function, are certainly precarious. This object is too large for our summary view of insanity; but it is too important to be passed over; and a glance at some aspects of it which seem important in practice may suggest others which cannot be remarked upon.

To appreciate the significance of alternative personalities, we must understand something about subconsciousness. In particular, we must note that, though the mind is not conscious of it, the brain may carry out a synthetic, a constructive process. That happens daily. The child, for example, acquires the elements of things—the yellowness, the softness, the smell, the shape of an orange, let us say—as separate; but his brain compounds them into a whole. And every one of active mind knows how things resolve themselves in sleep. You go to bed with two and two in your head, so to speak, and in the morning they have become four. The same thing happens when the mind entirely leaves a subject for a while and returns to it again. In the interval the subject has developed: that is, subconsciousness has been busy with it and the salient features of the thing stand out clearer than before. A man, for instance, who spends his days in the city and returns each evening to music, finds that, while his mind has been busy in the city, his music-brain has been making progress; and next day again it appears that, while his mind had been wrapt in music, his city-brain had solved some of his business difficulties. But these developments may not be all pleasant, and they may occur in relation to things which the mind has not attended to for years. The revival of long-past images in a new and compound form is common in primitive intelligences. By some such process we must probably explain the adoption by the insane of an entirely unexpected personality. The young mind perhaps was impressed by some Bible character or by some dramatic personation, and, years after, when normal personality subsides, the patient awakes to the fact that he is the prophet Daniel, or a brother of Prince Charlie, or Solomon himself, or perhaps King Lear. And when that happens, the patient does not have to think how such an one would feel and what he would say or do. The brain has made a character-

study for him and already knows the part. Far more commonly, however, the character to which the reduced consciousness attaches is one familiar, at least in its essentials, to the mind. It is a vice, for example, of hosts of idle minds to revel in day-dreams of an attractive form. Factory hands, we are told, are much addicted to the dissipation of resorting to a fancy-self, a glorified self, who perhaps lives in a castle, with many horses and untold servants, or who has wardrobefuls of fine dresses, countless admirers, and unlimited opportunity of romantic adventure; and those who have studied the mental habits of that class tell us that these people revert to this fancy-self when they have nothing else to think about, and, day after day, imagine themselves in their romantic character, enjoying a fancy-life in ever-varying circumstances. If these things be true, we have here an explanation of the assumption by the brain, when normal personality wanes, of a grandiose mode of consciousness and of a name which expresses it. For historical characters, long since dead, are not the most popular in insanity. The most commonly assumed identities are living celebrities and their relations, and nearly always aristocratic celebrities whose names are an emblem of rank and power. Many persons who pass for religious are victims of a like kind of vice. Their religious phase does not bind their whole life together, but is dissociated from general activities and is often almost entirely sensuous. It is a phase to which men and women resort when they wish "to forget the world," and then they languish in florid imagination and in unbridled feeling, very much as if under the influence of a narcotic. Such dissociations perhaps explain some of the religiose forms of insanity. Compare also the love-dreams of some minds, fed on sentimental novelettes; also the adventure-dreams of message-boys who gorge themselves on "penny dreadfuls."

Still more intelligible are those cases of alternative consciousness of which the two phases correspond to two actual stages in the life-history of the patient. Last year such a case came within my knowledge—a German colonel who spent his latter days in England. After an apoplexy he became aphasic and forgetful, subject, as these paralytic cases often are, to emotional excesses, and continually semi-delirious. Some days he was in a German phase, speaking German nearly all the time, and recalling German people and incidents. Other days he spoke English almost exclusively, and his reminiscences referred chiefly to English experiences. In his less pronounced phases, his disordered consciousness embraced both German and English images. Of such varieties, L. Bruce's[1] case is a good sample. His patient appeared to alternate between a right-sided and a left-sided celebration, with states also in which both hemispheres were active. At all events the man was in one phase an alert, mischievous maniac, whose sensibilities were keen, who spoke English, used the right hand freely and wrote, not very clearly, from left to right; and in another phase he was demented and dull, manifested unmistakable impairments of sensibility, spoke a kind of Welsh gibberish, carried his right hand usually in his pocket, but used his left freely, and wrote with his left hand in mirror-fashion. He also had ambidextrous phases whose mental state was a mixture of the English and Welsh modes. But the dissociation was so complete that in his pronounced Welsh phase he entirely forgot the facts of his English phase, and *vice versa*, though his English conciousness retained facts of its own phase.

But there are still other cases which cannot be explained as the revival of actual stages of a life's history. Many cases have been recorded of alternation between the real personality and that of some one else. All that we can surmise about such cases is that the brain had received vivid impressions of that other self, had subconsciously compounded them, and assumed the new personality at times and for a brief period to revert again, after a sleep perhaps, to the modes of activity of the real self. And, short of the assumption of a new identity, the loss of the real identity is very common in disordered minds. Many patients complain that they feel they are not themselves, and when they speak it is some one else's voice and not even their own opinions which we hear, or that the things they do are not their own actions but those of an interloper.

All such impairments of normal identity are peculiarly liable to occur in persons whose activities are too often dissociated. Stable brains are brains in which one mode of consciousness prevails and to which all other activities are subordinate. A brain which entertains diverse interests and pursuits not correlated by any unifying idea or feeling is by nature unstable, and an abrupt failure is apt to occur when one phase of the mind includes a conscience which will not condone the pursuits followed in the other phase. Tandem-driving is a precarious mode of progression when the tracer wants to be leader and the leader disapproves.

Insane Defects of Inhibition.—Sane inhibition is obviously impaired in all acute insanities. Melancholia or mania supervenes only when normal personality fails, and that implies that the patient's sane will is lost, though he may manifest determination or impulsiveness in disorderly directions. In Stupor, as we have seen, the expressional functions are profoundly disordered, the muscular or projective elements in life suffer great impairment, and anergia prevails. It may be that the will in its more

[1] *Brain*, vol. lxix.

central phases remains; that the patient is *trying* to behave sanely though the resistance in the ideo-motor and Rolandic mechanisms offers an insuperable barrier to the transmission of his dictates. We cannot say. But there are conditions in which inhibition is primarily impaired without any great interference with the rest of the mind such as we can appreciate. The best cases in point are those characterised by *Aboulia*, which is a simple anergia not usually so gross as to demand asylum treatment. The patient in the first instance simply fails to do the things which he has intended to do. That phase is common in a great many minor disorders, notably in those of alcoholic origin. But a deeper reduction of volition occurs in true Aboulia, when the patient suffers a loss of energy sufficient even for automatic acts and habits. And it is at the point of change from one kind of activity to another that the failure occurs. The affection is one which does not allow of interruption of the actual mode of consciousness of the moment. In the morning, when the patient ought to get up, he just stays on in bed; at bedtime he does not undress; he sits at table and cannot begin to eat; out for a walk he cannot turn to come home; he will sit for hours and not offer a remark. And all the time the mind is not profoundly affected. The patient is aware of his disability, and can, when once he starts, converse freely about it. But the risk is that, if rigorous treatment is not adopted—enforced regularity of hours, enforced hygiene, and enforced distraction from introspectiveness—the patient will become further disabled. Aboulia may go on to stupor—a systematic progress of the disease outwards and downwards towards the muscles. Or it may take a more central course and develop into *Folie de doute* or Swithering Insanity. Then the patient is liable at any moment, however, inopportune, to stop and wonder whether he ought to go on. That is, his intelligence is becoming involved, and a vicious habit is established in which the patient fails to do things, wonders why, wonders how she can, wonders whether she should, and still does not do them. This condition is, in a sense, a confusional insanity; but it is a confusion of impulses rather than of ideas. As in ideational confusion, the conspicuous feature is an excess of secondary suggestions. Its symbol should be a mark of interrogation. The patient can take nothing for granted, but questions the propriety or the advisability or the cause or the result of everything she is about to say or to do. As was said, the primary fact in such cases is the loss of common sense.

A distinctive feature of this insane indecision is to be found in the subjects to which it refers. It is not about important affairs—not about proposals of marriage, or a declaration of war, the offer of an appointment, or the investment of a fortune. In swithering insanity the patient hesitates as to which stocking to put on first, which garter, what boots to wear, which hat, whether to put the left or the right foot on the first of a flight of steps, whether to step on the lines of a pavement or to avoid them, and as to a hundred other things which likely do not matter a straw. In other words, the patient is beset by trivial questions which should have remained entirely beneath her notice. All the petty things of life should occur subconsciously, but in swithering insanity the trifles of subconsciousness confront the patient at every step. The condition is not without resemblance to Abasia Astasia. Let the physician try for a quarter of an hour during a walk to attend entirely to the art of walking—how one leg is to pass the other, exactly where each foot should fall, etc.—and he will understand how subversive of sanity it is to fetch up from subconsciousness the multitude of things that ought to lie there.

Or again, in impairments of inhibition, the mode of consciousness may be the obverse of *Folie de doute*, and the patient manifest an insane *Rhythmism* or automatism. Rhythmism rarely occurs conspicuously except as a concomitant of other and grave disorders. Its characteristic is that the brain continues to repeat a given movement indefinitely. Many patients, for example, go on repeating a single word or phrase indefinitely (verbigeration) without content or significance; or, if you ask them several questions, will give the same answer to them all. Or Rhythmism may manifest itself in what in the sane would be called habit-spasms, which have so interesting a relation with chorea. Stereotyped recurrent grimaces may occur, or the patient may go through a gesture again and again. More commonly, insane poses and attitudes and gaits insist upon constant reiteration, of which the most common, and a very unpleasant one, is a swaying motion of the trunk and head backwards and forwards. Kahlbaum described as an entity a series of symptoms to which he gave the name Katatonia, in which rhythmism and cataleptoid signs were prominent, but the condition is not now regarded as a specific or systematic affection. (Rhythmism, the repetitive mode of activity, characterises many of the insane. Insanity tends to habit. Note also the relationship of resistiveness—a revulsion against interference with the attitude or occupation of the moment, and of aboulia—an inability to stop doing one thing and to proceed to the next. Rhythmism also characterises obsessions.)

Insane Impulsiveness.—The affections previously described are essentially significant of a reduction of inhibition—a negative lesion. In others, however, though inhibition is often impaired, we have to suppose some positive

activities of an excessive kind, some hypersensibility to specified forms of stimulation. Such excesses of reaction are common in chronic insanities, and it is interesting to note that these are often reversions to childish proclivities —impulses to break glass, to throw down walls, to scratch smooth surfaces, to tear, and break and spoil things. Many of the insane cannot see anything worth destroying without an impulsion to destroy it. Biologically considered, such destructiveness is difficult to explain except as a form of mischievous playfulness which becomes manifest when disease has obliterated the acquired respect for property. Less harmful and more amusing is the temptation to touch and handle everything that catches the eye, to which some of the insane are prone. Either in sensory mania or in chronic insanity the patient may reach out to touch everything he passes that is bright or of an attractive form. It is impossible to keep up a flower garden to which the insane are admitted.

A far more important class of impulses, however, is that which includes insane proclivities to crimes and to vices. There cannot be any doubt that immorality is often determined by a morbid sensibility to vicious indulgences—a constitutional excess of temptation,—though we need not, on that account, condone all such failings or expect that they will be "cured" by measures which are milder and which provide less of a deterrent against vice and less of an incentive to virtue than such measures as we adopt with sane offenders. For the understanding of these insane temptations the physician may contrast them with the phobias, some of which we have considered. In the case of these, the patient suffers an abnormal abhorrence or dread of certain things, while in impulsiveness the balance of feeling is all the other way. The subjects of phobias are not obviously the subjects of insane impulses, but we might compile a considerable list to emphasise the antithesis. Thus—pyrophobia (fear of fire) and pyromania (the impulse to set afire); gynephobia and anthrophobia or nymphophobia (sexual fear) and nymphomania; dipsophobia (fanatic abstinence, cf. hydrophobia) and dipsomania; kleptophobia and kleptomania; agoraphobia and agoramania (the impulse to take to the open fields); claustrophobia and claustromania (the impulse to take to human burrows); acro- or batophobia (the fear of high places) and acro- or batomania (impulse to precipitation).

Violent impulses often beset the degenerate, and, as was said, we cannot call a man degenerate who is of sound moral nature. Degenerates are often moral imbeciles. That is to say, they fail to appreciate goodness, to understand the value of it, even to recognise it when it is exhibited to them, and still more are incapable of being good. Over and above that general immorality they often experience a violent impulse to commit ordinary crimes, and sometimes are impelled to commit horrors over which they gloat. It then becomes a medico-legal point to determine whether the impulse, with or without other symptoms, constituted an insanity. Short of such cases as come into the police court, however, the physician often has to determine the sanity or insanity of a private patient who has been guilty of some misdemeanour. He can only do so by a systematic investigation of the patient's mental functions, including the moral sense, by an inquiry into the heredity of the case, and by noting such physical facts as might betoken or occasion a debased or unstable character. Further, the circumstances of the case must be carefully weighed; for we must remember that in many cases the patient has been subjected to what might be a great temptation even to a sane mind.

Impulses of nearly every kind are common in the insane. We may here advert to the danger of *mutilations* which mostly occur in melancholia, in paranoia, and in general paralysis. Sometimes a patient feels impelled to mutilate a neighbour, sometimes himself. Three cases occur to me, all of them patients who were living at home. One of them, who subsequently developed a climacteric melancholia, was beset by the impulse to amputate the penis of her paralytic husband, but refrained, and was sent to an asylum. The second, a puerperal case, had to leave home because of the dread of mutilating her child; and that is a common kind of danger in all puerperal insanity. The third, a lactational melancholic, tried to cut off her child's hands with a table-knife.

Many patients pull their hair out, or try to gouge out their eyes, or lacerate their flesh, or amputate a finger. Attempts at castration or at amputation of the penis are very common in men who entertain sexual delusions or who are greatly tormented by sexual impulses, and often signify masturbation. These self-mutilations are very unintelligible from the point of view of evolution. We have to remember, however, that very few people are content to leave their bodies as nature made them, and that barbaric people resorted to many devices to alter their shapes; also that mutilations, especially of sex-organs, were associated with many unenlightened religions.

In the insane, impulses are very often the result of *obsession*—an insane mental process which is closely related to delusions and hallucinations, but which is best regarded in association with impulse. An obsession is a persistent and recurring idea which forces itself on the mind and will not be denied. Obsessions cannot always be distinguished from hallucinations. The latter are automatic activities in some part of the sensory tracts which spring an unreal perception on mind. Obsessions are

also automatic activities. They do not require any external stimulus, though, as is the case with hallucinations, they may be determined by circumstances. A sense of silence, of loneliness, far-off, faint sounds, or sights or smells may occasion them. Both hallucinations and obsessions occur most seldom when the mind is most stimulated by normal sensorial activities. Their prime character, then, is that they are in some degree fulminating or impulsive. They are sensory or ideational activities which correspond more or less to epileptiform motor symptoms. In mania, all manner of suggestions are sprung upon the mind. In obsession it is the same idea, again and again and again. There is another parallelism, then, between an obsession (*sensori-ideational*) and say verbigeration (word repetition, *motor*). Both are rhythmic or repetitive. Hallucinations of hearing are not obsessions, but *a hallucination*, such as that of the words "You are a fool," might become an obsession. We limit the term to ideas. Any recurrent hallucination which conveys an idea may become an obsession. But not always, for, if the patient hardly attended to the words, there would be no obsession. That is the last quality of obsession—that it invades the personality. The patient accepts the idea as his, recognises it as part of his mind. Obsessions, then, are ideas which are occasioned by automatic cerebral activities, which are repeated with great frequency, and which invade the personality. And there is no end to the variety of them. Many of them are so silly and so irrelevant that we cannot understand their obtaining a foothold in the mind. Others come very near home, so to speak, have an intimate personal reference. But they defy any attempt at classification, for, as Macpherson has it, "there are as many forms of obsession as there are of thought." For the practical physician, however, it is useful to note (1) those which are, in a technical sense, indifferent, and which only bother the patient by their persistence; (2) those which excite well-marked emotion, and in these the emotion is usually grief or fear (cf. the phobias); and (3) those which suggest some specific line of conduct and tend to become impulses. Of the last, the obsession which besets the true dipsomaniac and the transition into the corresponding impulse is specially considered elsewhere (*see* "Dipsomania") and may be taken as exemplifying the class.

Insane impulses, whether preceded by obsession or not, are too manifold to be considered in detail. An impulse, in the technical sense, has these characteristics:—That it does not require objective stimuli though it may have an appropriate occasion; that is the persistent (rhythmic); that the patient is impotent to resist it; that satiety follows upon the indulgence of it. The most important perhaps are homicidal or suicidal. Patients who run amuck are often the victims of impulse—feel an irresistible craving to kill people. Sometimes suicide is an almost unrelated impulse, occurring in patients who are not melancholic and not delusional. Dipsomania, kleptomania, nymphomania, pyromania, are other important forms of impulse. As every one knows, these impulses may occur without any other obvious disorder of the mind. But anything that can enter into the mind of man as a thing to say or to do may become an impulse in the strict sense. A patient may be beset by the impulse to count, to turn the hands of clocks, to unscrew all screws, to look for pins, to put things in the fire, to pour out water, etc. etc. etc.

The diseases of the will which appeal most to the public are, of course, those which result in vice or in crime. Drunkenness is treated of in the article "Alcoholism," and dipsomania is specially described. That separateness of treatment illustrates two aspects of all insane vice. There is the insane impulse, as in dipsomania, due to defective organisation and the development of an excessive susceptibility; and there is education or habit, as in drunkenness, which fosters the original sin. In *sexual perversion* and in *sexual excess* both these factors play a part. In sodomy, in masturbation, and in other such vices, we have often to note a defective organisation of a negative form, comparable to aphobia. Femininism in men, masculinism in women, denote a constitutional aversion to normal love, as well as a perverted desire for the abnormal. Much more important, on the whole, for it is now very common, especially in women, is a simple absence of sexual enjoyment. It is a serious question whether modern culture implies a failure of instincts and capacities essential to the race. Still more does the importance of the subject appear when we realise how often a sexual distaste spoils the happy fellowship of man and wife, and eventually precludes the possibility of a successful home. The fact of such a difficulty is revealed so often in the consulting-room that one is compelled to regard it as a large factor in the troubles of present-day society. The defects of a too prudish upbringing are in some measure to blame for the vice. Both sexual perverts and married inverts would to some extent be prevented if youths grew up in the belief that sexual desires and feelings were not at all improper, much less wicked, but both necessary and wholesome. In proper form and in appropriate circumstances sexual activities are as essential as are eating and drinking—which is a commonplace that is implicitly denied in our present-day upbringing. Excepting in those who will not, or should not, marry, it has become a duty to develop connubial, maternal, and paternal capacity. For the rest society suffers perhaps for a too lenient view of sexual offences. There are very few cases in which punishment of them would not be beneficial. But a wise view of sexual offences

is not to be insisted on until wise views upon inoffensive sexual functions have been learned.

Sometimes a task scarcely less delicate falls to the physician who is asked to determine the nature of a supposed case of kleptomania. He will in any case have to choose between describing the patient as dishonest or as insane. The culprit might usually be left to say; for most cases are both. Most kleptomaniacs are women, perhaps for want of business education. The abeyance of sufficient motive, some weak-mindedness, and an approach to the insane diathesis, are corroborative. In pure kleptomania there is well-marked impulse, but a large number of spurious cases are rather the subjects of doubtful habit—of thought, if not of deed—and have encouraged the wrong idea. In this connection it is interesting to note the relation of hoarding to pilfering. Misers and other "collectors" are prone to pilfering. I was consulted recently about a young woman who, in less excusable circumstances than most, took money belonging to others and hoarded it. When a girl she was unusually fond of money and frequently appropriated coins and hid them in strange places, mostly in books. Treatment is difficult. All cases require moral discipline, as well as physical bracing. Punishment of ordinary kinds will do harm in genuine cases when there is some degree of tumultuous impulse perhaps amounting to an epileptic equivalent. But patients who encourage wrong impulse may be deterred by suitable corrections. Pilfering from comrades is severely punished in the army and is comparatively rare.

Systematised Insane Purpose.—We need only glance at this form of mental disorder. It is not a recognised form of insanity, and the practitioner will seldom have to treat a private case. But it will be well if he recognises it as an insanity, and forms an opinion as to how to deal with it. For the patients involved will sooner or later become the subjects of legislation or of provision by our courts.

A systematised insane purpose is a deliberate intention to achieve some definite end by insane means. It differs from impulse in that it is a deliberate and abiding purpose, not a passing crave; and it is usually the expression of a conviction which may or may not be a delusion. A paranoiac, for example—and degenerates and paranoiacs are most subject to this form of madness—may be convinced that some one is his arch-enemy, and may consequently plot murder and deliberately set about to achieve it. Many paranoiacs conceive projects which are much more insane than the delusions of which they are the outcome. Vindictiveness, or at least the supposed redress of wrongs, is the motive in nearly all such cases. Very often there is a real and serious grievance. The insanity lies in the method adopted to achieve the desired result. And we must judge of such an insanity by taking into account the tradition and customs of the time. Sanity is the capacity of the individual to appreciate, and to regulate his conduct according to, the accepted standards of his day. An old-world Corsican, for example, whose family had suffered great injury or insult, who undertook a vendetta and slew the offender's grandson, would not, in his day, have been necessarily insane. But such an one who to-morrow revenged a real or supposed injury by stabbing his victim in the streets of London, must needs either be hanged as a murderer or detained as a dangerous lunatic. The motives of such insane revenge are usually either personal, or religious, or political. Insane purposes are often entertained by weak-minded persons who have a personal grievance. Others conceive that God or Allah, or some other deity, desires the death of certain people, and set about to accomplish it. But political crimes are those with which we are most familiar. The revolt of the Boxers in China and their intention to massacre Europeans would be an insanity in point, did they not represent just about the level of Chinese thinking and of Chinese morals. Nihilism is of this nature. And perhaps the best examples are to be found in anarchists, members of a civilised and law-abiding community, who set out to put wrong things right by insane and often violent means. From the point of view of mental science, political crimes are in no way justified by the fact that the patient entertains lofty sentiments about the rights of man any more than we would overlook the insanity of a patient who slept with his wife and family in the open fields because he entertained some enlightened views about tubercle. Much has been made of the love of notoriety as an important factor in political crimes. Its importance is probably exaggerated. The essential facts in such cases are that the intelligence is weak, so that the patient cannot think straight or see the various sides of his question; that he is possessed by disordered feelings of resentment which leave no room for justice and mercy towards innocent individuals; and especially that he has suffered an arrest of development which is obvious in his disability to believe in, and have patience with, constitutional methods of redress. In any case, the only possible treatment of such social insanities is by stern preventive measures. If anarchists and such-like criminals were quietly consigned to a criminal lunatic asylum their craving for notoriety would soon dwindle; and if this form of insanity were generally recognised, if it were understood that any one who advocated insane methods was *ipso facto* a lunatic, and a fit subject for asylum treatment, the mad crimes with which the world is so often horrified would materially diminish.

Dementia.—All uncured insanities which are not terminated by early death result in some

degree of dementia. After a few years, the longer an insanity lasts the more purposeless and silly the mind becomes. Primary dementia has been referred to, and, in some cases, a consecutive dementia, greatly resembling it in onset and course, follows soon upon an acute melancholia, stupor, or mania. Indeed, as was said, the acute insanity often seems to be a mere incident in the mental dissolution which is imminent. For that is what dementia is—a reduction of mental faculties; and it must be distinguished from states of imbecility in which the mental functions have never properly developed. A thorough dementia is a premature and rapid dotage. The various functions gradually dwindle, and the patient apparently does not think, is incapable of emotion, has no purposes, but is merely a creature of habits. Then his motor functions suffer; no thorough dement is capable of any work which requires skill: the sensibilities become blunt; the trophic functions are impaired, and the organic reflexes; and the patient slowly dies.

A great many dementias, however, are not thorough, but partial. The distinction is so obvious that one is inclined to think that every case of rapid and thorough dementia is an idiopathic dissolution, a premature senility, ushered in perhaps by an acute insanity. But the great majority of dements are only partially demented. The disease is limited, and it is not progressive. It is impossible to classify these secondary degenerations, for any one mental function may manifest the chief failure or a disproportionate survival, and in every case there is some general reduction, as well as an extreme degree of particular reduction. But these partial dements are interesting because they, more than most of the actually insane states, illustrate a reversion to primitive modes of consciousness. Every asylum, for example, can produce cases of insane constructiveness—patients who spend their days in harmless but quite puerile activity. Others are insanely predatory, or shamelessly acquisitive, and annex useful and useless things as indiscriminately as a magpie. Others spend hours daily in barbaric forms of self-adornment, or revel in vain exhibitions of themselves with ludicrous affectation. The sexual and the domestic instincts possess many partial dements, and these patients delight in promiscuous and often harmless flirtation, or in the furnishing of their rooms with ridiculous and lavish ornaments. In short, a partial dement may manifest any form of absurd and futile activity. For a general dementia is always notable in these cases—a decay of sane personality and a disability for serious and profitable occupation.

To the last, then, insanity is a systematic disorder. Subconsciousness is arranged and orderly, true to itself, though perhaps not suitable to a polite environment. In acute insanities, when sane personality is for the time being in abeyance, and in slow or partial dementia when it is for ever lost, the instinctive feelings and ideas and impulses of which subconsciousness consists, and which are a survival of ancestral or of personal experience, take its place and determine insane character. Yet is that character always intelligible and systematic—as orderly as the consciousness of a child, of a savage, or of a lower animal, and as certainly represented in nervous organisation.

VI. Index of Etiological Varieties of Insanity

Dr. Urquhart[1] has purposely omitted an account of the forms which insanity often takes appropriate to the particular causes which he describes. Several of the etiological varieties are described in special articles. But it may be useful to suggest others. Although these are not all so constant as to be recognised as typical insanities, the practitioner may find it useful to know what forms mental disorders usually take, given certain etiological conditions.

(1) Idiopathic.—Imbecility (*see* "Mental Deficiency"); Degeneracy; Primary Dementia (*see above*); Paranoia (*q.v.*).

(2) Occasioned at critical periods of life.—Adolescence (*q.v.*); Pregnancy (*q.v.*); the Climacteric (*q.v.*); Senility (*q.v.*).

Insanity of Maturity.—Occurs when the patient incurs heavy responsibilities. Anxious melancholia, often proceeding to acute mania.

(3) *Persuaded Insanity.*—(*a*) Folie à Deux—A deluded patient persuades another to believe him; separation usually cures the second. (*b*) Folie à Plusieurs—Several persons may follow an insane lead. Babcock (*Amer. Journ. of Insan.* April 1895) quotes a good case where a household and several neighbours acquiesced in a systematised delusion of persecution. (*c*) Epidemic Insanity—Excitement, superstition, odd behaviour, frequently signs like saltatorial spasms.

(4) Toxæmic and Dyscrasic Disorders:—

(*a*) Stimulants, narcotics, and grain poisons. *Alcoholism* (*q.v.*); *Chloralism*, *Cocainism*, *Morphinism* (*see* "Morphinomania"); *Ergotism.*—Gastro-intestinal and vascular disorders, varied mental symptoms often like general paralysis because of motor affections. *Pellagra.*—Stupidity, irritability, deep depression, delusions of fear. *Plumbism.*—Headache, insomnia, bad dreams, remitting delirium, or progressive delusional insanity.

(*b*) Specific Infections. *Typhoid*, etc.—Delirious and acute excited insanity in early stages, slow, depressed insanity often with motor signs in convalescent stage. *Influenza.*—The same, but motor signs often accentuated so as to resemble general paralysis. *Syphilis.*—Insane fear with delusions of syphilis when none exists,

[1] Page 442.

delusional melancholia in early stages, in later stages (tertiary and onwards) progressive dementia, or delusional suspicion and impulsiveness, or epileptic insanity, or tabetic insanity of anomalous forms, or resembling insanity of deprivation, or pseudo-general paralysis, or general paralysis. *Tuberculosis.* — In active phthisis, suspiciousness, moroseness, quarrels, fickle melancholia with mild delusions, or with hysteria, or with stupor, and, later, acute mania, or suspicious melancholia often with refusal of food and impulsiveness, with masking of physical signs, and, if not recovered, ending in fitful, asocial, querulous, semi-dementia; in slow tuberculosis, especially if abdominal, visceral melancholia. *Septicæmia.*—Acute, confused, delirious insanity, often recoverable, or ending in death by exhaustion, or going on to chronic mania with delusions and hallucinations (this includes some surgical cases and some puerperal cases).

(*c*) Constitutional Diseases. *Gout.*—In active gout, angry melancholia, or delirious mania (especially in over-use of sedatives), in "retrocessed gout," violent insanity perhaps epileptoid, in "suppressed gout," slow insanity often with hallucinations, in "undeveloped gout," neuralgic, neuritic, hysterical excitement, or moodiness. *Rheumatism.*—In acute fever, cf. "Typhoid," later a recoverable mania, melancholia rarer, or a slow amnesic, anergic insanity. *Diabetes.*—Melancholia with delusions of fear in persistent diabetes, or acute mania with temporary disappearance of glycosurea. *Uræmia.*—Fearful melancholia, great dread, exacerbations of violent excitement, impulsiveness.

(*d*) Affections of Nervous System. *Shock* (includes some puerperal and some surgical cases). —Confused, excited insanity with affection of memory, fancifulness, and perhaps hallucinations. *Traumatism.*—Moodiness, suspicion, outbursts of excitement, amnesia, motor signs, altered reaction to drugs. *Sunstroke.*—Neurasthenia, debility, motor or sensory signs, altered reaction to drugs, headache, melancholia and hypochondriasis, oftener exaltation and resemblance to general paralysis. *Neurasthenia.*—Various insanities. *Hysteria.*—Vain mania, often erotic. *Epilepsy* (*q.v.*). *Chorea* (*q.v.*). *Apoplexy.*—In later stages, excited insanity, amnesia, great proneness to laughing and crying, impulsiveness, violence, dementia. *Deprivations* (of hearing, sight, etc., and should include *aphasia* and some other lost movements).—Vexation, self-consciousness, dread, irritability, confusion, suspicion, excitement, delusion; melancholia rarer. *General Paralysis* (*q.v.*). *Tabes.*—General paralysis, or, not infrequently, pseudo-general paralysis, or anomalous insanity with chronic exaltation and exacerbations of violence.

(*e*) Disorders of Digestive System. *Paradigestion.* — Various insanities. *Dyspepsia.*—Melancholia with hypochondriasis. *New Growths.*—Visceral melancholia, with delusions of suspicion.

(*f*) Disorders of Respiratory System. *Phthisis* (above). *Asthma.*—Recurrent mania or chronic mania. *Pneumonia.*—Cf. "Typhoid."

(*g*) Disorders of Heart, Vessels, and Blood. *Cardiac Disease.*—Melancholia with delusions of fear; in aortic disease, violence and variability commoner; dementia early. *Atheroma.*—Excited suspicions, melancholia, dementia. *Aneurysm.* — Visceral delusions. *Anœmia.*—Melancholia in men and in mature women; in young people, stupor or fickle, fanciful, excitable insanity with erotic and hysterical symptoms. *Myxœdema* (see "Thyroid"). *Cretinism* (*q.v.*).

(*h*) Affections of Reproductive and Genital Organs. *Ovarian and Uterine Disease.*—Recurrent insanity, delusions of sexual misfortune with melancholia, or delusions of exalted marriage with mania, modification of secondary sexual characteristics (beard, etc.); (delusions of a disagreeable kind often attach to some one person in the neighbourhood). *Functional Excess.*—Curable stupor, or progressive dementia. *Prostatic Disease* and *Stricture.*—Vexation, irritability, dread, excitement, sometimes going on to delusions of suspicion.

Insanity, General Treatment of.

THE GENERAL TREATMENT OF INSANITY.—Disease is known by the failure of function to satisfy the need of the organism. Treatment of disease is the endeavour to readjust the balance between function and need. There are two methods by which this balance may be restored. The impaired function may recover its efficiency, so that it again suffices to satisfy the need, or the need may be reduced until the impaired function is sufficient to satisfy it.

The restoration of function by artificial interference is seldom possible. Instant restoration, when it can be effected, is almost exclusively the privilege of the surgeon. He can instantly reopen the lumen of a hollow viscus whose obliteration prevented the passage of the visceral content. He can remove the tumour or evacuate the abscess, or cut the constriction which is interfering with function. He can tie the artery whose wound allows of the escape of that blood

which it ought to contain. To the physician the direct restoration of function is less often possible, and when possible has rarely the dramatic suddenness and completeness which are achieved by the successful effort of the surgeon. The disturbing agent whose removal he attempts is rarely of the gross mechanical nature that so often has to be dealt with by the surgeon. Often it is some subtle poison to which he possesses no antidote; often it is some diffused and microscopic degradation of tissue whose restoration, even if the degradation can be recognised, is beyond the resources of his art. Only when he possesses an antidote, only when he can neutralise the poison by the administration of some appropriate drug, as by mercury or iodide of potassium in syphilis, or by an appropriate antitoxin in a specific fever, can he pursue the direct method of restoration of function in the former class. In the latter he is altogether powerless to pursue this method. All that he can do is to reduce the need to the level of the impaired function. When the aeration of the blood is impaired by disease of the respiratory organs or of the heart, he reduces the need for aeration by forbidding exercise. When the digesting power of the stomach is impaired, he reduces the need for digestion by reducing the quantity of food, or by administering food easy of digestion or already partly digested, or by administering digestive agents with the food. When the kidney fails in its power of eliminating nitrogenised waste products, he diminishes the need for such elimination by diminishing the ingestion of nitrogenised food. When writing is rendered impossible by writer's cramp, he diminishes the need for writing by ordering the use of a typewriter. When the function of a joint is impaired by inflammation, he keeps the joint at rest. In any case he reduces the need for the function as far as possible down to the capacity of the function impaired, and thus restores the balance between function and need.

It is evident that this second mode of restoring the balance is a poor substitute for the first. The restoration of the impaired function to its normal standard is a true *restitutio ad integrum*, and brings the organism back to its full measure of capacity. The reduction of the demand of the organism down to the level that the impaired function can reach is at best a makeshift, and an acknowledgment of our impotence to do better; but this second mode of restoring the balance is not without its influence on the first. The reduction of demand upon an impaired function influences the restoration of the function in both a favourable and an unfavourable direction. On the one hand, the reduction of demand upon the function, especially if the reduction can be pushed to actual cessation, sets, in the one case a part, and in the other the whole, of the anabolic process free to work at the restoration of the damaged structure, which otherwise would be engaged in the double process of restoring structure, and at the same time actuating function, a combination that is rarely within its capacity. When the hull of a ship is damaged, she must be brought into dock for repair; when her engines or her boilers are outworn, they cannot be made good while she is on active service; and similarly the tissues of the human body cannot undergo repair so long as full functional activity is demanded of them. Only during intermittence of function can redintegration progress; and hence those tissues in which waste is most rapid and function most exhausting, need the frequent functional cessation of sleep to keep them in repair. The reduction of demand upon an impaired function is therefore a most important condition for the restoration of the function, and is in very many cases the chief, in many cases the sole, means at our command of bringing about the restoration of the function. On the other hand, no facts in physiology are better established than that function tends to increase with the demand that is made upon it, and that prolonged diminution of demand is followed by diminution of function. From the arm of the blacksmith to the verbal memory of the actor, we see how demand increases function; from the leg of the omnibus-driver to the intelligence of the soldier, we see how lack of demand leads to cessation of function. Hence we are taught that while on the one hand, as soon as a function is impaired by disease, the demand upon it should be immediately reduced to the lowest possible level, yet on the other hand, as soon as the process of repair is well on the way, the demand should be cautiously increased, so as to lead forward and assist in the re-establishment of the function.

Applying these principles to the special case of insanity, we find that the treatment of that, as of all other maladies, is twofold. The main end in view in every case must be the restoration of function to its full capacity, and as a means to this end the demand upon the function must be reduced, at first to or below the level of the capacity of the impaired function. As and when improvement of function takes place, the demand upon it must be cautiously raised, and kept always just a little in advance of its capacity, so as to lure it by degrees to wider flights and stronger efforts. Even if no improvement appears to indicate the period at which increasing demands may be made, still the increase of function must, after a time, be called for, and persistently called for, such demand being commonly the only means at our disposal of eliciting an improvement in function.

Seeing that of these two modes or aims of treatment of insanity the second is always possible, is always imperative, and the mode of effecting it usually clear and simple, it may be dealt with first; while as the other can be

pursued only tentatively, by groping in the dark, and is often, in the present state of our knowledge, not possible at all, its consideration may be deferred.

The Reduction of Function.—The function which in insanity is disordered and deteriorated is that of the supreme regions of the nervous system, by which conduct is actuated, by which the metabolism of all parts of the body, both absolutely and relatively to each other, is regulated, and which is accompanied by states and changes of consciousness. Of these functions the first alone is open to our direct interference. The others we can modify indirectly only. The need for that adjustment, by intelligent acts, of the organism to its surroundings, which is termed conduct, we can diminish almost *ad libitum* by furnishing a set of surroundings so completely preadjusted by artificial means to the needs of the organism, that voluntary adjustment can be dispensed with. All the more intelligent phases of conduct, and, if necessary, all phases of conduct but the most rudimentary, can be supplied by artificial substitutes, and the highest nerve regions, thus relieved of their functions, can be left at rest to deal with the disturbing agent that is acting on them, to recuperate their deteriorated structure, untrammelled by the need of simultaneously carrying on their function.

In all cases of insanity, or of defect or disorder of mind or of conduct, which, though of the same nature as insanity, would not be called by so serious a title—in all cases of such disorder, whether incipient or established, whether mild or aggravated, the first need is the need of rest, of reduction or abolition of the demand upon the disordered function. The recognition of this need has sometimes led to the pursuit of an exaggerated course, and has been interpreted to mean that every such patient, whatever the form or degree of his malady, must be deprived as completely as possible of all opportunity of conduct, by being put to bed. This is as intelligent a mode of treatment as to put to bed every patient with a broken bone, regardless of whether the bone broken is that of a leg or an arm or a finger. "He physic's use doth quite mistake, who physic takes for physic's sake." If those parts of the nervous system, which regulate the most complicated relations of the organism with its surroundings, are unequal to the demand upon them, and cannot regulate their relations efficiently, that is a reason for reducing or removing the demand upon them; but if inferior portions are still capable of carrying on their functions with efficiency, they may still be permitted to do so, even though their superiors must be relieved. If the apparatus by which a man carries on his business is disordered, it is right and necessary that he should be relieved from the opportunity of carrying on his business; but it is no more necessary that he should therefore be relieved from the opportunity of walking about and amusing himself than that he should be relieved of the opportunity of voluntarily emptying his bladder. If every insane person is to receive what is humorously termed "hospital treatment," that is to say, is to be kept in bed, the treatment is not consistent unless he is also relieved by a catheter and fed with a stomach-pump.

The difficulty of suspending or reducing the call upon function in insanity lies in the differences between insanity and other maladies. If a man suffers from any bodily malady, he seeks, his conduct not being disordered, the advice of some competent authority, who gives the advice and leaves to the patient the responsibility of acting upon it. But in insanity it is conduct itself which is disordered, together with mind, and hence in the first place the patient very often does not know that he is suffering from any malady; and in the second, if he do know, he is often unable, from the disorder to which his conduct is subject, to take the course with regard to it which even he himself may see to be expedient. For this reason it is often necessary in insanity to act without the consent, and even in opposition to the wishes, of the patient, and to deprive him forcibly of those opportunities, which he will not willingly relinquish, of exercising his disordered faculties.

It is often necessary, but it is far from being, as is sometimes supposed, always necessary. In the case of minors, they can be taken away from the school or the college or the course of studies in which the excessive demand upon the immature brain has caused the break-down. In the case of the adult, he can often be persuaded into, nay, will often himself suggest, a temporary retirement from active employment, and a season of fallow, when he feels that his capacity is no longer equal to the demand upon it; and in mild degrees of the malady, and in these only, such measures are sufficient; but not only are such measures impracticable in the case of those who will not submit to them; they are also often inefficient even in the case of those who will. A man who knows himself unfit for business, who feels his incapacity to follow his profession, may voluntarily intern himself in an asylum, and go from bad to worse until it becomes necessary that he shall be certified and compulsorily detained; and from the day that this is done he begins to improve and makes steady progress to recovery. It seems as if the mere responsibility for determining his own course of conduct were too heavy for him to bear; that he was weighed down by the uncertainty as to whether he was doing right; and that only when this responsibility was entirely removed by his compulsory seclusion from the ranks of those *sui juris*, only when he could feel that he was relieved of all responsibility for the regulation of his own conduct, was obtained that complete

quiescence of the highest nerve regions which rendered recuperation possible.

To take over by force the regulation of a man's conduct, is a proceeding which lies entirely outside the province of the physician. Such a proceeding is *ipso facto* a menace to every member of the community. It is one which is not to be countenanced except upon the strongest grounds of expediency, and it is one, moreover, which the community will not and ought not to permit without the sanction of its own accredited representative, given under specified conditions laid down by itself. These conditions vary very widely in different communities, and even in the several divisions of these islands they exhibit a surprising diversity. Broadly, it may be stated that the formalities whose observance is required in depriving a "person of unsound mind" of the regulation of his own affairs are divisible into two classes, those which refer to the deprivation of his management of his means of livelihood, and those which refer to the deprivation of his management of the rest of his relations with his surroundings. His means of livelihood are called in English law his estate, while the maintenance of due relations between himself and his surroundings in other respects—between himself and physical dangers, between himself and his family, between himself and the community in all respects other than his means of livelihood—is regarded as peculiarly the management of himself. With the formalities which are necessary for the deprivation from a person of the management of his estate, the physician is not directly concerned, and they need not be considered here, further than by mention of the fact that, under the English law, a person may be deprived of the power of managing his estate, while he may be at the same time left in undisturbed possession of the management of all his other relations with surrounding circumstances.

The formalities necessary for depriving a person of the management of these residual relations differ, as has been said, in the three divisions of the United Kingdom, and they differ in each division, moreover, according to the degree to which the deprivation is carried. For degrees of deprivation are made, and a person may be deprived of the management of more or fewer of his relations with his surroundings, or, as we may say more briefly, of his affairs, according as it appears expedient. It is very much to be regretted that in the laws of England only the grosser degrees of incapacity can be dealt with by deprivation of power. Every year, nay, every month, and almost every week, occur cases in which a goodly estate is dissipated, an honoured name is besmirched, an innocent family is brought to scandal and to poverty, by the prodigality, the vice, the folly of some unworthy scion who is incapable of prudence and rectitude, but whose incapability is not of such a nature as to enable his relatives to put the provisions of the Lunacy Acts into force for their own protection. There is not an alienist physician, there is not a family solicitor in large practice, nay, there is not an intelligent reader of the law and police news in the daily papers, who is not aware of cases of this kind, in which, if not scandal, at least impoverishment could have been averted, had the excellent provisions of the French *conseil de famille* been in force in this country. However, our concern here is with the law not as it ought to, but as it does exist, and with this preface we will proceed to state it as it stands.

The legal provisions under which a person can be deprived of the regulation of his own affairs differ, first, according as his defect in regulating power is congenital or not; and, second, according to the amount of his property.

England and Wales.—An *idiot or imbecile* from birth or from an early age may be placed under control, and detained in an institution registered under the Idiots Act, upon the certificate of one medical practitioner, accompanied by a statement made by the parent or guardian of the patient or by the person who undertakes towards him the duty of a parent or guardian (Idiots Act, 1886, sect. 4). As these documents are of a very simple character, need no comment, and are obtainable at any registered institution for idiots, there is no need to introduce them here.

A lunatic *wandering at large* must be apprehended by the police and brought before a justice, who will send the patient to the county or borough asylum of the district. Either before or after he is so sent his friends may obtain control of him by making application, in the one case to the magistrate, in the other to the visitors of the asylum and to the Commissioners in Lunacy.

A lunatic *who is not under proper care and control, or who is cruelly treated or neglected* by those who have charge of him, must be reported by the police on oath to a justice, who will proceed as directed in the Act.

A lunatic *who is a pauper* must be reported to the relieving officer of the district by the union medical officer of his district within three days of the medical officer obtaining knowledge that the pauper patient is or is deemed to be a lunatic.

For a lunatic *who is not a pauper* the procedure is various.

The patient may, if he is willing to do so, and if he is competent to form a judgment as to the expediency of doing so, enter a licensed house as a voluntary boarder, by previously obtaining the consent of one of the licensers—of a commissioner in the case of a metropolitan licensed house, or a visiting justice in the case of a provincial licensed house; or he may enter as a voluntary boarder a registered hospital without any previous consent other than that of the

manager of the institution. In any case he must not be so insane as to be certifiable.

A Bill is now before Parliament authorising the temporary admission into private care of a lunatic whose insanity is not confirmed, under a single medical certificate and without any further authority. This provision is adopted from the Scotch law, which see.

If the patient is certifiable, and if his malady is "confirmed" (*see infra*), there are three modes by which his detention under care and treatment may be legalised: the judicial reception order, the urgency order, and the inquisition.

The judicial reception order is obtained from a justice specially appointed under the Act, or from a stipendiary magistrate, or from a County Court judge, by means of a petition from a relative of the patient, accompanied by a statement of particulars and supported by two medical certificates. There is no need to give here the form of the medical certificate, which is sufficiently well known, nor is any accomplishment needed for its correct execution beyond ordinary care and ordinary intelligence. It is nevertheless remarkable how few certificates are correctly executed. The obvious precaution of referring to all the marginal notes and complying with the instructions that they contain is usually neglected, as is the other obvious precaution of reading it carefully through after it is made. The consequence of this neglect is that certificates, which are discreditable to their signators, are very frequently sent to the commissioners, by whom they are returned for the correction of palpable blunders which never ought to have been made. Apart from the form of the certificate, which can be made in due order by the exercise of the most ordinary care, the substance demands attention. The object of the certificate is to satisfy the mind of the judicial authority to whom the petition is presented, that the patient is of unsound mind, and a proper person to be detained under care and treatment. It should, therefore, contain such evidence of these facts as shall carry conviction to the mind of a person who has never seen the patient and knows nothing about him. Too often the medical practitioner looks upon himself as the only person who has to be satisfied of these conditions, and embodies in his certificate little more than an assurance that he is satisfied. This is a very erroneous view. What is required by the justice is not the conclusion at which the medical practitioner has arrived, but the grounds of this conclusion. The terms of the certificate itself are quite sufficient to indicate what is required. They require the practitioner to state that he has arrived at the conclusion that the patient is of unsound mind, etc., and that he is a proper person to be taken charge of and detained under care and treatment; and further, that "I have formed this conclusion on the following grounds, viz.: facts indicating insanity observed by myself at the time of examination," and "facts communicated to me by others." It would be difficult to put into more explicit terms the requirement of the law, and yet this requirement is frequently ignored. Very commonly, under the head of "facts indicating insanity," is inserted a diagnosis, and still more commonly is inserted an opinion which the certifier mistakes for an observed fact. The only "facts," rightly so called, which can properly be stated in a certificate are the sayings, doings, and in some cases the appearance of the patient. Seeing that the certificate states that the patient is of unsound mind, the certifier frequently falls into the error of enumerating states of mind as "facts indicating insanity." He will say that the patient is depressed, that he suffers from delirium, that he thinks this, and feels that, and believes the other. But these are not "facts observed at the time of examination," or at any other time. They are opinions. They are inferences drawn from the sayings and doings of the patient; and it is these sayings and doings, and not the inferences from them, that are in fact observed, and that alone may rightly be included in the certificate. If the act or statement of the patient is manifestly insane; if, for instance, he hands his interlocutor a scrap of newspaper on which is written a cheque for a million pounds; or if he knocks off his visitor's hat and jumps upon it, then no more than the bare recital of the fact is needed. But if the act or statement is only conditionally insane; if, that is to say, it cannot be known to be insane without a knowledge of the circumstances of the patient, then so much of these circumstances as suffice to establish the insanity of the act or statement must be embodied in the certificate. If, for instance, the patient states that he is ruined, or that his leg is broken, or that his wife has deserted him,—statements which are only conditionally insane,—then the practitioner must satisfy himself, first, that the statement is untrue; second, that the patient cannot be convinced of its untruth; and must state these qualifying facts, as well as the primary fact of the statement itself, in the certificate.

The disadvantage of the procedure by judicial reception order is that, requiring as it does the concurrence of several—at least four—persons, it necessarily occupies time, and sometimes the necessity of taking out of a person's own hands the control of his affairs is so instant and importunate that this time cannot be given. For these cases is provided the procedure by *urgency* order, under which it is lawful to detain a patient for a limited time upon the order of the relative, accompanied by a medical certificate, not only of his insanity and of the evidence of it, but likewise of the reasons which render it expedient that he should be forthwith placed under care

without waiting for a judicial reception order. The reason for urgency may be either the welfare of the patient or the public safety. When a patient is detained under an urgency order he must be released at the end of seven days unless in the meantime a petition for a judicial reception order has been presented to a judicial authority; but if within seven days such a petition has been presented, then he may be detained under the urgency order until that petition has been disposed of. There is at the present time a measure before Parliament which, if it becomes law, will reduce the period of independent validity of the urgency order from seven days to four.

The inquisition is a measure to which recourse is had only when the property of the lunatic is considerable, or when he demands an inquiry into his sanity with a view to his release. It is of the nature of a trial at law, and the function of the physician with regard to it is merely to give evidence when called upon. An inquisition results, if the petitioner is wholly successful, in the appointment of a committee of the person and of a committee of the estate of the lunatic, who are responsible to the court for the proper performance of their duties. Each committee may consist of one or more persons, and the same person or persons may act in both capacities. It is competent to the court to find that the lunatic is unfit to manage his affairs, but fit to manage himself, and in that event no committee of his person is appointed, and his personal liberty is not restricted.

Scotland.—Idiots and imbeciles under 18 years of age may be received into training schools in Scotland without certification or other legal formality. In practice, however, pauper children are invariably sent thither under the same formalities as are used in the case of adults sent to other institutions for lunatics, with the addition that the consent of a commissioner must be first obtained. The reason for this procedure is that when so sent only is the school entitled for each child to a 4s. grant. When above the age of 18, and when sent to institutions other than training schools, idiots and imbeciles are in Scotland classed as lunatics, and are subject to the same formalities.

Insane persons whose malady is not confirmed may be placed in private care for a period not exceeding six months, under the special certificate of one medical practitioner, and without other formality.

The ordinary procedure in pauper and non-pauper lunatics is the same, and requires a petition to the sheriff accompanied by a statement of particulars and the certificate of two medical practitioners. In the case of a pauper the petitioner is the inspector of poor.

If the case is urgent, a certificate of emergency by a single medical practitioner suffices to authorise the detention of the patient for three days. This certificate must be accompanied by a request, from the person in the position of petitioner, to the superintendent of the asylum, to receive the patient.

In addition to these provisions, the Scottish law provides a process termed *interdiction*, which has no counterpart in English, though it resembles certain proceedings in French law, with which it has a common origin in the *jus civile*. Interdiction is a legal restraint or incompetence fixed upon persons who are prodigal, or are too easily influenced by others, and disables them from alienating their property without the consent of guardians, who are called interdictors. Interdiction may be voluntarily entered into by the interdictee, or may be compulsorily enforced upon him by the Supreme Court, and in the latter case the decree of the court may be granted either upon the initiative of the relatives of the *prodigus*, or upon the initiative of the court itself, founded upon its own observation of the conduct of a party to a suit before it.

The proceedings of cognition and curatory are analogous to the inquisition in English law, from which, however, they have wide differences. If successful, they result in the appointment of a committee, or, as he is called, a curator, to administer the estate of the lunatic; but there is no functionary corresponding to the committee of the person of the lunatic.

Ireland.—The formalities differ according as the patient is to be taken to a district asylum on the one hand, or to a licensed or unlicensed private house on the other.

Pauper patients who are not dangerous are admitted *into a district asylum* upon four documents:—1. Declaration before a magistrate stating that the patient is insane and destitute, and giving names, etc., of two relatives. 2. Certificate of a magistrate and a clergyman, or poor law guardian, that they have personally inquired into the case. 3. One medical certificate. 4. An engagement by the applicant to remove the patient when called on.

Paying patients who are not dangerous require five documents to authorise their admission into a district asylum:—1. Declaration before a magistrate that the patient has not sufficient means to pay for support in a private asylum, and has no friend who can do so, and stating how long the patient has been resident in the country. 2. Certificate of a magistrate and a clergyman, that the case has been investigated. 3. One medical certificate signed by two medical practitioners. 4. An engagement to remove, together with an engagement to pay a specified sum. 5. The sanction of an inspector of lunatics.

The proceedings above described are so cumbrous that in practice the great majority of patients in the district asylums of Ireland are admitted as *dangerous lunatics*. Any person

may be apprehended "under circumstances denoting derangement of mind and an intention of committing an indictable offence," and removed by warrant of the Lord-Lieutenant from gaol to an asylum.

Into *licensed houses, charitable institutions, and single care*, patients are admitted upon the following documents:—1. An order by a relative or connection of the lunatic. 2. A medical certificate of two medical practitioners.

In cases of urgency the signature of a single medical practitioner is sufficient, provided that a second be added within fourteen days of the first.

Finally, the proceedings under *inquisition*, which are practically the same as in England.

The Advisability of Asylum Treatment.—From the foregoing account of the methods of procedure in force for the legal deprivation from persons of unsound mind of the management of their affairs, it will appear that several degrees and modes of deprivation are recognised and sanctioned by the law, and the first question that will present itself to the practitioner, when he has determined that his patient is of unsound mind, will be, Which of these degrees and modes is proper to be adopted in this case? Ought the patients to go to an institution, or to be placed in private care? and in either case ought he to be persuaded to go, and ought his remaining there to depend upon his own free will as it is influenced by moral suasion, or ought he to be deprived of his liberty by due process of law?

The decision must depend upon several very diverse considerations, of which the chief are—The nature of the malady, the age of the patient, and his means.

1. *The Nature of the Malady.*—A patient who is definitely suicidal or acutely maniacal ought to be at once sent under certificates to an institution. To this rule there is no exception. It is only in an institution that the structure and appliances of the building are specially arranged for the accommodation of such patients. It is only in institutions that the windows, the appliances for lighting and heating, the staircases, the water-closets, the bedsteads, and a score of other arrangements, are specially adapted to obviate the suicidal or dangerous or destructive proclivities of the patient; and although it is, of course, within the competence of persons of sufficient wealth to adapt the structure of their houses and the contents of their houses in a similar way, it is not competent to them to effect these alterations with the necessary speed to secure the immediate safety of their relative. Moreover, such patients need, if not the continuous services of a medical attendant, at any rate the continuous presence of a medical attendant within call; and this is scarcely to be obtained at a moment's notice in a private house. Even in the case of a man of unbounded wealth, who could secure the services of an unlimited number of experienced attendants of all grades, the element of time would always necessitate the immediate removal of the patient to an institution as a first step. The rule admits, therefore, of no exception. But the question immediately arises, How are such patients to be recognised? With respect to the maniacal patient there is no difficulty. The degree of his excitement, the outrageousness of his acts, at once place the matter beyond doubt. But with the suicidal patient the difficulty is often great. A definite attempt at suicide, of course, settles the matter. After that there can be no doubt. But it by no means follows that a melancholic patient who has made no attempt, and no verbal reference to suicide, has yet not formed a determination to take his life on the first opportunity; and it often tests to the uttermost the diagnostic power of the most experienced expert to determine in any given case whether the tendency exists or no. As a rule, the non-expert greatly under-estimates the likelihood of a suicidal tendency. The expert, on the other hand, tends rather to over-estimate it. But then the latter has probably seen such disastrous results from the under-estimation that he deliberately chooses to err upon the safe side. Whenever melancholia is decided, is pronounced, and whenever mania is acute, and is unattended with exaltation, a suicidal tendency should be strongly suspected, nay, should be "conclusively presumed," and measures should be taken accordingly. It is not said that where these features are not present the suicidal tendency will be absent, but that where they are present it may be presumed.

Where neither suicidal tendency nor acute mania is present, the question of treatment in an institution will depend upon the amount of control that it is necessary to exercise. Where the amount of control needed is not great; where the patient does not need very vigilant supervision; where his disorder of conduct is not of such a nature as to imperil his own safety or that of others; is not displayed in sexual matters; is not such as to render him very conspicuous; is displayed only at intervals, and on occasions that can be foreseen and provided against; then he may be properly treated in private care. But when the conditions are the reverse of these he ought to be placed in an institution. There the amount of control that can be exercised, and that ordinarily is exercised, is much greater than in private care, and therefore that is the proper destination of patients for whom much control is needed. For this reason patients with delusions of persecution are unsuitable for private care. Every such patient is a potential homicide, and requires an amount of control that need not be very strict, but that must be continuous; and continuity of control can be secured much more certainly in an insti-

tution than in private care. Generally, the graver the disorder of conduct, the more pronounced the malady, the more suitable is the case for treatment in an institution; and *vice versa.*

The *age* of the patient is a factor of some importance in coming to a decision. We are very reluctant to send a young girl or a lad to an institution for lunatics if such a course can be avoided. So long as the savour of the "madhouse" clings to these institutions in the mind of the public, so long as any remnant of that semi-superstitious horror remains with which the ignorant public is apt to regard them, so long we must avoid, if it be possible to avoid, sending to them young people who have all their careers before them, and whose future lives may be more seriously affected by the reputation of having been in a lunatic asylum than by the reputation of having been unsound in mind.

Lastly, the *means* of the patient is a factor of considerable importance. Private care is, as a rule, a more expensive mode of control than care in an institution, and when the amount of control that is needed is great the disproportion is correspondingly great. A patient who needs continuous supervision both by day and by night, will in private care need the services of at least two attendants, and if the supervision is prolonged, of three; and attendants whose services are temporarily engaged, as in private care, demand much higher wages than those whose engagements are permanent. In an institution the same patient would not need the exclusive services of these attendants, and each attendant would be less costly; so that if expense is a consideration the institution is to be chosen.

As these are the main factors in determining whether the patient shall be sent to an institution or treated in private care, so are they the main factors in determining whether resort shall be had to legal process for restricting the power of the patient over his own conduct.

If the case be one of mere dementia no such resort is needed, unless for reasons of expense or convenience it is determined to send the patient to an institution, and the sending must be legalised. Mere dements are already deprived by their malady of the control of their conduct; and no intervention is necessary to do what the disease has already done. All that they require is the same supervision and nursing that is required by children of equivalent intelligence. In the case of minors whose conduct needs more active control, certification can usually be avoided, since they are not yet fully *sui juris*, and the authority of the parent to control their conduct does not, except in severe cases, need to be reinforced by the special provision of the lunacy law. If the parent chooses to say to his child, "I have made arrangements for you to leave home and to be placed in the care of So and so, to whose authority you must submit," the child has no alternative but to obey. When an adult is but slightly affected by unsoundness of mind, and is himself aware of his unsoundness, and desirous of placing himself under treatment, then of course he may make whatever arrangements he pleases for relinquishing the control of portions of his affairs. He may give power of attorney to whomsoever he pleases to deal with his estate, and he may take what advice seems to him judicious and desirable as to the disposal of other departments of his conduct. He may place himself in private care or in an institution as seems best.

Advice is often given, especially by neurologists who have no special knowledge of insanity, but are frequently consulted with regard to it, that the patient should travel, either by himself, or with a friend, or under the care of an attendant, medical or other. Such advice is rarely judicious. The object of suggesting a change of surroundings is to liberate the patient from the necessity of regulating his conduct in certain departments; to reduce the call upon function. But in travelling, while if he relinquishes the care of his estate, the call upon function is diminished in this direction, it is manifestly increased in others. The constant change of surroundings brings of necessity the constant need of changes of conduct in adaptation to the changes of surroundings, and the relief that is obtained in one direction is nullified by the increased demand that is made in others; so that we find in practice that in the early stages of mental disorder travelling is not beneficial. The cases in which it is most commonly advised are cases of slight melancholia, and the advice is given from the analogy of the relief from care that is gained by jaded men of business from a holiday with change of scene. But the two cases are not analogous. The jaded man of business has lost the elasticity of his spirits from a too complete engrossment in the cares of business,—from the circumstances in which he acts. His depression is due to, and proportionate to the stress of circumstances upon him, and when relieved from the stress of circumstances he recovers. But with the melancholic it is otherwise. However much the stress of circumstances may have contributed to the establishment of his malady, it is, when established, independent of his circumstances. The definition of melancholia is that it is a state of misery that is not justified by the circumstances of the patient; and therefore the mere relief from these circumstances is not sufficient to remove it. What the melancholic, with his conviction of his own incompetence, needs, is circumstances that make no demand upon his incompetence; and these circumstances are not to be found in travelling. Let him leave home by all means; but let him go to some one spot and there remain, in surroundings in which demands upon his faculty of regulating his own conduct are minimised.

The greatest mistake of all is made when the melancholic is recommended a sea-voyage. Here he is placed in circumstances of dismal monotony, with the temptation to suicide constantly pressed upon his attention. What wonder if he often succumbs?

Regulation of Control.—Having complied with the legal formalities, and released the patient from the obligation of regulating for himself the more elaborate portions at any rate of his conduct, the next question in treatment that presents itself is, to what extent this release is to be given or this control to be exercised. This must depend upon the depth to which conduct is disordered. If only a superficial flake is peeled off from the upper strata of his capabilities, — if, for instance, as sometimes happens, his conduct is disordered in certain social relations only, — if his conduct say towards the members of his family, or towards members of the other sex, or towards religious ceremonies, or towards children, or with respect to legal proceedings, is the only region in which his conduct is disordered—then there is no need to exercise control over him except with regard to the particular relation disordered. There is no reason why persons whose disorder of conduct is limited to such particular divisions as those above specified should be deprived, for instance, of the management of their "estate," nor of other aspects of their affairs. The limitation of disorder of conduct and the limitation of control which logically follows upon it, are explicitly recognised both in English and Scotch law by provisions which allow the patient to retain the management of his other affairs, while he is deprived wholly or partly of the management of his estate; and there is no reason, save the difficulty of formulating in words the department of conduct disordered, and of precisely limiting the control to that department, why the principle should not be generally applied in statutory enactments. But if not applied in law, it can and ought to be applied in practice, and the principle upon which control should be exercised over lunatics should be, in all save acute and recent cases, that the control should extend over that region of conduct only which is disordered. This principle is already followed to some extent in practice, but it is followed partially, haltingly, and with no true perception of its full application. All patients who are detained as lunatics are not controlled in the same degree. Some are allowed their liberty on parole, others are not; some are allowed the restricted use of money, others are not; some are allowed to regulate their own costume in accordance with their own taste, others are not; most are allowed to take their own food in such quantities as they desire, others are forcibly fed; and other distinctions are in practice. But in most institutions and in every large institution there is a certain fixed maximum of freedom which no patient is allowed to exceed, and this maximum is far too low, and is fixed without regard to the considerable class of patients whose activities need restricting in one direction only, and in different directions for each.

To this rule, that conduct should not be controlled further than it is disordered, an exemption has been made. In all acute and recent cases the control must be very complete; and this for several reasons. In the first place, until the patient has been under observation for some time it is not known with certainty how far conduct is in fact disordered. In the second place, it is in acute and recent cases that a principle already alluded to applies, viz. that the demand upon function shall be withdrawn very completely so as to leave the metabolic process at full liberty to restore the damaged structure. The validity of this exception is fully borne out in practice. It is found that in acute and recent cases the mere removal from home and compulsory detention among strangers has of itself, without further measures, a distinct ameliorative effect; and it is a well-established fact, known to all who have care of the insane, that during the early days of their malady the visits of relatives and friends with whom they are in habits of social intercourse has a detrimental effect. The beneficial influence of detention under control is often ascribed to the mere change of surroundings of every kind which it usually involves, and it is probable that a part of this beneficial influence is correctly so ascribed; but it is probable also that in the presence of near relatives and customary friends the patient feels that the control over him is relaxed, and that he may venture upon a degree of self-assertion which in their absence he would not aspire to. Be the explanation what it may, the fact is indisputable that in recent and acute cases of insanity the mere removal from home and the detention under care have of themselves a beneficial effect, an effect which is at once neutralised for the time being by the visit of near relatives. Fortunately the ill effect of those visits does not usually endure. Sometimes the melancholic who has poured out his woes to his wife and has wept copiously over his imaginary troubles, will cheer up on the instant of her departure and join in amusements as heartily as he was doing when her visit was announced. More often it takes him a few hours to get over it, but I cannot say that I have observed any permanent ill effect.

It is in cases in which the disorder or defect of conduct is deepest, in which the most fundamental and primary departments of conduct are affected, that the control must be most complete. Where the primary instinct of self-preservation is absent, or is reversed; in the deeper forms of dementia, in which almost all forms of

conduct are lost, and the patient is reduced to the infantile condition; when he is incapable of preserving himself from simple mechanical dangers; when he cannot be trusted alone in a room for fear he should fall out of his chair into the fire, or stuff into his mouth any uneatable substance—coal, rag, or paper—within his reach; when, like an infant, he is inattentive to the state of distension of his rectum and bladder, and passes his excretion as he sits; or when, instead of using his activity to preserve his life, he endeavours with all his resources to put an end to it; in either case the control over him must be of the most minute and vigilant character. But the two cases are manifestly different. The one may be left to the nurse and the general physician. The other needs the utmost exertions of the most skilled attendants on the insane. The only chance of preventing a suicidal patient from carrying out his intention is incessant and minute vigilance. Even incessant vigilance is not always successful, but it is the only means at our disposal, and it must be employed.

When the case is no longer recent; when time has been given for any acute process of disorder in the brain to subside, and the stage has arrived for attempting to restore the deficient function, and to re-educate the patient into normal modes of conduct, then control must be gradually lessened, and the patient must be encouraged and stimulated to exercise those departments of conduct of which he has been hitherto deprived. So long as the bones of a broken arm are ununited, so long must the arm be kept at rest in a splint, and its function suspended. So long as the valves of the heart are the subject of acute inflammation, so long must the function of the heart be minimised by keeping the patient in absolute rest. But as soon as the bones are united we encourage the patient to overcome the stiffness of his fingers by using them; as soon as the cardiac inflammation has subsided we encourage the patient by graduated exercise to strengthen the muscle of his heart; and as soon as the acuteness of the outbreak of insanity passes away, we should encourage the patient to resume, tentatively and intermittently at first, but with gradually increasing frequency and completeness, the control of his own relations with his surroundings. It is at this time that the skill of the alienist is tested to its utmost capacity.

Other General and Special Treatment.—Leaving now the question of the regulation of conduct by external coercion, we enter on the consideration of the second branch of treatment, and ask what means we have of acting, by ingesta and in other ways, upon the disordered cerebral processes so as to bring about their restoration to the normal. We find that our knowledge of such means is much on a par with the knowledge that Voltaire attributed to metaphysicians, —"fort peu de chose." Something, however, we have gained.

Under normal circumstances the great restorer of the function of the highest regions of the brain is sleep. Towards the end of every day sets in an exhaustion of these regions which, if pushed to extreme, exhibits itself in disorder. No one is as active, as alert, as mentally competent, after a sleepless night as after a night of sound sleep; and the persistent deprivation of sleep of itself leads to great disorder. To this we add the knowledge that in all cases of acute and recent insanity sleep has recently been defective, and thus we gain a clear and indisputable indication for treatment in all such cases. Measures must be taken for the induction of sleep. And the propriety of the indication for treatment is shown by the result. Until sleep is induced improvement does not take place. When sleep is induced, improvement does, as a rule, occur. How, then, is sleep to be induced? The obvious reply is by the administration of an hypnotic drug, and for many years the routine administration of opium in recent cases of insanity was followed, and the natural consequences were such that a reaction, as unreasonable as the practice, set in against the administration of all hypnotics whatever, and a most pernicious maxim gained currency that "chemical restraint is as bad as mechanical restraint." It is difficult to say which part of this question-begging statement is the most to be condemned, whether the false analogy between sleep and restraint, or the implication that mechanical restraint is, *ipso facto*, and in all cases, bad. If a patient is sleepless; and if I have a drug the administration of which will produce sleep; and if after the sleep so produced the patient is better, is more normal both in mind and conduct; am I to be precluded from the administration because of the stupid and unjustifiable statement that I am restraining my patient by chemical means? Such slavery to a form of words would be unworthy of the most narrow-minded pedant that ever exercised his degraded ingenuity in Westminster Hall. But is it a fact that sleep so produced is ever beneficial? I affirm with the utmost certainty that it is; and I go farther, and say that by the administration of drugs we have the power in certain cases of converting a furious maniac into a comparatively sane man; of reducing his maniacal fury down to mere restlessness, and even to tranquillity; and of freeing his mind, temporarily at any rate, from the delusions to which it was subject. This may be "chemical restraint," if we please to call it so, and if it be, let us be heartily thankful that the resources of modern pharmacology have placed such a means of restraint at our service. Of course it will be said that I advocate the indiscriminate and excessive use of hypnotics in every case of insanity. How far this accusa-

tion is justified will be seen from the following remarks :—

In inducing sleep our first object must be to place the patient in those physiological conditions in which sleep normally occurs. What are these conditions? If we knew them all, and could reproduce them, we could induce sleep without administering any drug at all; but, as we know only some of them, this is not always possible, although, since the missing condition may be one of those that we do know of and are able to supply, it sometimes happens that we are able by mere attention to hygiene to induce sleep without administering any drug. The two conditions that we can with the greatest confidence associate with the induction of sleep are fatigue and repletion. Other things being equal, the greater the previous exertion and the greater the fatigue, the greater the tendency to sleep; and not only is there a very definite synchronism between the absorption of food and the induction of sleep, but deficiency of food is inimical to sleep. When therefore we have to deal with a patient who suffers from sleeplessness, it is important to secure for him a healthy degree of fatigue by inducing him to take a healthy amount of muscular exertion, and especially of exertion in the open air; and it is far more important to secure for him the absorption of food. In the majority of cases of acute insanity that come under treatment the patient is suffering from starvation. He has either refused or neglected to take food in sufficient quantity, and his sleeplessness has the same source as that of the man who has had to go to bed supperless. Nothing, not even acute pain, is so efficient a cause of vigil as an empty belly. Even in the torments of toothache and of gout snatches of sleep are possible, but the hungry man does not sleep even for a moment. Hence our first measure in the treatment of acute insanity, accompanied, as it always is, by insomnia, is to give a copious meal of some easily assimilated food. Slops and stimulants, concentrated foods and meat extracts, are not what is needed. What is needed is a bellyful of easily assimilated food ;—Benger's food, revalenta, arrowroot, rice and milk, or better, if the patient will take it, and his digestion can cope with it, beefsteak and potatoes, bread and butter —anything, in short, that is both nourishing and bulky. Not seldom this measure and this alone is enough to secure a sound and long sleep to a patient who has scarcely slept at all for weeks. And in any case the treatment is right. The waste of the superior cerebral regions on which the insanity depends may be of such a character that it cannot recover even when copious supplies of pabulum are presented to it; but whatever its character, it certainly cannot be made good without material to make it good; and hence, in all cases of acute and recent insanity, copious feeding is required. Young people sometimes come under care with the history that their appetite is good, that they have been eating pretty well. But in such cases the statement must be received with a qualification. The amount of food that they have taken may have been sufficient for the needs of a mature adult; but the mature adult, the parent who gives us the account, does not realise how greatly the need for food of the adolescent and the immature adult exceeds that of the mature adult. The lad of 18 or 20 needs at least three times as much food as the man of 45 or 50, and what is sufficient, judged by the standard of the one, is very insufficient for the needs of the other. On every account the administration of abundance of food is the first necessity in the treatment of acute insanity, especially in the young.

But it is not enough that food should be administered. It must be absorbed. And in acute insanity, especially in melancholia, the digestive functions are almost always disordered. Even in the sane, the depression that accompanies dyspepsia is sufficiently notorious. In morbid depression dyspepsia, or other disorder of the processes of the gastro-intestinal canal, is scarcely ever absent. This, then, must be the next care of the alienist physician, and here again the value of regulated exercise and a well-ordered diet will soon make itself apparent.

If food alone is insufficient to induce sleep, and commonly it is not by itself sufficient, attention must be paid to the circumstances under which sleep is sought, which may include some that are preventing sleep. Every one knows the greater difficulty of sleeping in the sweltering heat of summer as compared with that of sleeping in cold winter nights; and many a case of insomnia may be relieved by the removal of two or three superfluous blankets. Coldness of the feet is a very effectual preventive of sleep, and the insertion of a hot bottle in the bed will often render the administration of a drug unnecessary. The importance of quiet need not be insisted on, and of all the complaints made by patients of ill-treatment in lunatic asylums, none has seemed to me more bitter, nor better founded, than that which refers to the flashing of a lantern in the eyes of sleepers by the night watchman, a practice which is highly inimical to sleep, and ought not to be permitted.

Supposing, however, that when attention has been paid to all these matters of hygiene, and that the patient is still sleepless, resort must be had to hypnotic drugs, and of these we have a large number to choose from. The particular drug chosen will vary with the character of the malady, but in all cases the best practice is to begin with a large dose, and to give this dose time to act, rather than to give repeated small doses at frequent intervals. When a patient is

in a state of very acute mania, raving and raging and ramping, there is no drug that approaches in rapidity and efficiency of action to hyoscin. It is said to be uncertain in its action, and occasionally to produce dangerous effects, but these I have never seen, although I have used it largely, and in much larger doses than are customary; and I am inclined to believe that where it has had ill effects it has either been improperly prepared, or has been administered in larger doses than the administrator was aware of. In such a case as has been mentioned, my practice is to give one-fiftieth of a grain hypodermically or one-twenty-fifth of a grain by the mouth, and usually the patient is asleep in five minutes, and wakes after from six to ten hours without an unpleasant symptom. The effect of the drug, even when administered hypodermically, is sometimes delayed; I have known it to take no effect whatever for as much as two hours; but it must not be repeated for at least twelve hours, and I have never known it to fail. It may be that thus to produce a tranquil sleep almost immediately in a patient who was howling, roaring, dancing, struggling, smashing, biting and kicking—it may be that to produce in him an instant and tranquil sleep, from which he awakes quiet and comparatively rational, or, as has happened in one case under my care, from which he awakes completely and permanently sane—it may be that this is chemical restraint, and if it be, let us be thankful that we have such a means of restraint at our command.

When the mania is less acute and of a more lasting character, and especially in the excitement of general paralysis, no drug has the efficacy of sulphonal, the action of which is somewhat peculiar. It is very insoluble, and has no hypnotic effect for some hours after its introduction into the stomach; on the other hand, its action, unlike that of hyoscin, is very enduring. After a sufficient dose of sulphonal a maniacal patient will not only obtain several hours' sleep, but after he awakes the drug will maintain its action, and the patient will be tranquil, but rather stupid, and apt to be drowsy for the whole of the following day. In other words, as sulphonal is slowly absorbed, so it is slowly eliminated; and when it is given by routine in daily doses it accumulates in the body and produces disastrous effects. Sulphonal should never, therefore, be given continuously. Its use should be alternated with that of other hypnotics, and even when not alternated it should not be given for more than three or four days in succession. In view of the delay in its absorption, it is always wise to combine, at any rate, the first dose of sulphonal with a less quantity of its congener trional, whose action is much more rapid and more transient. The trional puts the patient to sleep, and the sulphonal maintains the sleep. For the first night 30 or 40 grains of sulphonal may be combined with 20 of trional; and this may be followed the next afternoon by 30 grains of sulphonal, and the third or, at any rate, the fourth night, the patient will usually sleep without any drug.

Bromide of potassium, chloral hydrate, and opium with its component alkaloids, are little used as hypnotics in the treatment of the insane. Bromide of potassium has its use as a calmative in the mania which is associated with epilepsy, and when administered for this purpose is of little use in less than drachm doses. Chloral hydrate is not to be recommended except as a variant to be used during the intermission of other drugs. Opium is now perhaps as much neglected as, when our resources were more limited, it was used in excess. As a hypnotic, indeed, it has little value, but it often appears to have a distinct ameliorative effect in melancholia, especially in the melancholia of middle and advanced age, given regularly two or three times a day. Of course in thus administering opium or morphia we run great risk of establishing an opium or morphia habit, but this is a risk that must be run. The choice is between two evils—between leaving the patient in the most wretched condition in which a human being can be, or relieving him at a cost which is indeed great, but which both he and all to whom he is dear would be willing to pay for his relief. As a mere hypnotic the most appropriate drug in melancholia is paraldehyde.

Apart from the administration of food as an hypnotic, it is of very great importance upon other grounds in the treatment of insanity. Less important, perhaps, in the insanities of old age and middle life than in those of youth, it is yet important in all. As most cases of recent and acute insanity that come under care are found to have been for some time sleepless, so most of such cases are found to have been for some time underfed, and a copious supply of food is usually as important an element in their treatment as is the induction of sleep. Often it happens that the patient will not take food; more often that the food that he does take is not assimilated. There are patients who will not eat in company, or if they know that they are observed; and there are patients who will not eat alone. There are those who will not eat from conscientious scruples that they do not deserve it, or that it is not paid for, or that they are not entitled to it; and there are those who will not eat anything that they have not previously stolen. There are patients who take insane prejudices against this or that kind of food, and again patients to whom this or that kind of food alone is acceptable. By far the most important peculiarity is the refusal of food, and this may be of various

degrees of intensity. With some patients it is the first step only which costs, and after he has been compelled to take a spoonful he will eat the rest of his meal voluntarily. Another will take nothing voluntarily, but will allow himself to be spoon fed; others will need to be fed with the tube, but will take contentedly whatever is thus administered; while yet others will resist to the uttermost any attempt to feed them, and when at length they are fed will thrust their fingers into their throats and do their utmost to induce vomiting of the food given. Very many methods have been adopted of feeding those who refuse food, and an account of most of them may be found in Hack Tuke's *Dictionary of Psychological Medicine*. Practically the methods may be reduced to three—the spoon, the nasal tube, and the ordinary stomach tube. The spoon should be used where it can be used, and where it cannot the stomach tube is undoubtedly the best alternative. The numerous deaths that have occurred from pneumonia after the use of the nasal tube are alone sufficient to condemn it. A patient who refuses food is suicidal, and the introduction of food by the nasal tube gives him an opportunity of inhaling the food purposely if this do not occur by accident; or if the tube is passed sufficiently far to obviate this danger he can bring it forward into his mouth, bite off the end, and swallow it. In feeding by the stomach tube it is important to remember two precautions. The tube should be well softened in scalding water, and the food should be strained through a sieve so as to free it from lumps which would block the tube. No reliance can be placed upon the assurance of the cook that the food has been strained, and the operator should strain it himself or have a strainer inserted into the funnel. With regard to the nature of the food, the slops enumerated on a previous page are the most appropriate. Liebig's extract, Bovril, Valentine's meat juice, and other extracts can be added if it is thought expedient; but as their nutritive value is low, and as there is no palate to consider, there is not much object in doing so. More important is the administration of helps to digestion, of zymine, of pepsine, lacto-pepsin, etc.

In melancholia, and in acute insanity generally, there is almost always some disorder of function of the stomach and intestines, and this has to be considered and dealt with. Usually when the stomach tube is passed a few bubbles of air will escape from the stomach, and in some cases this air is horribly fœtid. When this is the case the stomach should be systematically washed out before the administration of food, and after its fœtid contents have been withdrawn it should be flooded with a solution of permanganate of potash or, better, of sanitas oil, which has a much more powerful deodorising effect, and to this treatment may be added the administration of β-naphthol or other intestinal disinfectant.

Apart from those that have been enumerated, our means of dealing with the morbid processes that underlie insanity are extremely limited, as indeed may be inferred from our ignorance of their nature. It is found, however, that there are certain morbid states of body which accompany certain forms or cases of insanity, and that the insanity fluctuates, recovers, and relapses, *pari passu*, with the bodily state. The connection of melancholia with gastro-intestinal disorder has already been referred to, and cases have been recorded of cystitis and other bodily maladies with which insanity coexisted and with which it varied. Hence it is incumbent in every case of insanity to investigate thoroughly the bodily health, and to treat any bodily disorder that may be discovered in the hope that with its improvement the mental state also may improve.

The opinion is more and more gaining ground that in a large proportion of cases of insanity the disturbing agent is a poison of some kind. In general paralysis, in acute delirious mania, in puerperal, alcoholic, and saturnine insanities this mode of origin exists without doubt; and true curative treatment of insanity will begin when our knowledge is sufficiently advanced to enable us to eliminate, destroy, or counteract these poisons before they had produced irretrievable structural damage. This knowledge we have not yet reached, and in the meantime our therapeutical measures are limited to treating whatever morbid process we can find that is amenable to treatment, and so, by inducing a general state of good health, to place the tissues of the individual in the most favourable state for eliminating or destroying the poison.

In view of this mode of the causation of insanity, it is upon organo-therapy and serum-therapy that our hopes are chiefly based of eventually treating insanity in a scientific manner, and the tentative efforts that have been made in this direction are not destitute of encouragement, though more than this cannot yet be said of them. The administration of thyroid extract has been tried in a large number of cases besides those of cretinism and myxœdema, in which its action is so marvellous, and in a few of these cases recovery has followed under circumstances that leave little doubt that the recovery was due to the administration of the extract; but no class of cases has yet been identified as specially amenable to this mode of treatment, and treatment by serum-therapy has not yet begun.

While many of the poisons that serve in the production of insanity are unknown, and while those that are known are not amenable to the action of any known antidote, there are some which, if we cannot deal with them when once

they have gained entrance to the body, we can at least arrest at the door and prevent from entering. We cannot undo the mischief that alcohol has done to the brain, but we can at least see to it that no more alcohol is imbibed, and so of lead, morphia, chloral, and other ingested poisons. In puerperal insanity, also, we can clear out the uterus, and counteract the septicity of its contents, which, in some cases at least, appears to be contributory to the insanity.

Dr. Haig has written much and strenuously upon the evil effect of an animal diet in bringing about uric-acidæmia and melancholia, and upon the advantages in this psychosis of regulating the diet and administering salicylates. There is no doubt that in melancholia the blood-pressure is high, but beyond this there is nothing in the majority of cases of melancholia to corroborate Dr. Haig's doctrine. It is very rarely that a case of melancholia is improved by the administration of salicylates, but here and there we do meet with a case, usually that of a portly, middle-aged man who has lived pretty freely, whose depression does appear to be favourably influenced by the administration of these compounds. The cases in which they are likely to be useful are those in which the urine, scanty and of high specific gravity during the day, is copious and watery in the night, and in which the patient has to get out of bed once or twice to pass water—cases, in short, of incipient renal disease. The combination of depression of spirits with these symptoms is not very common, in spite of all that Dr. Haig maintains, but when they are so combined the administration of salicylates has a very marked effect in improving both the one and the other.

There are certain phases of conduct exhibited by the insane which demand special notice with reference to their treatment.

Suicidal Tendency.—This is extremely common in acute insanity. There is a prevalent opinion among those whose experience of insanity is limited that mania and melancholia are entirely distinct forms of the malady, and that the tendency or impulsion to suicide occurs in the latter form only. This view is entirely erroneous. It is true that the deliberate intention to commit suicide is very often associated with melancholia, but it is also true that conduct of the most determined and persistent suicidal character is often exhibited in states of acute mania in which no other sign of depression is apparent, and in which there does not seem to be enough mind for the formation of deliberate intention. It is in such cases that refusal of food is most obstinate, and that impulsive rushes to the windows, attempts at strangulation, and so forth, are most common. For the prevention of suicide in these, as in other cases, there is but one method which has any chance of success, and that is the exercise of incessant vigilance. Under no circumstances, neither by night nor day, must the patient be left unwatched for a single moment. He must be accompanied in his goings out and his comings in. He must be taken to the closet and watched while there, and an attendant must sit by his bedside all night. And the attendant must be experienced in the ways of such patients, and fully impressed with the necessity of making the vigilance incessant. A patient whose head is under the bed-clothes is practically as well screened from observation as if he were in a room alone, and in such circumstances can stuff his mouth with a corner of the sheet, and so suffocate himself effectually, while his attendant sits by unsuspecting.

Masturbation is a very common habit in the insane of both sexes, and one that is very difficult to counteract. Those who are still sufficiently governed by the restraint of modesty to restrain from it while they are under observation should be kept under observation to the same extent as a suicidal patient, but many of those who are addicted to this habit own no such restraint. In them continuous and laborious employment is the most effectual remedy. They should be worked in the garden, the field, or the shop until they are dog-tired, kept up late, made to rise early, and lightly clothed at night. In those cases in which the habit is most fixed, the patients are as a rule too demented to be capable of any but the simplest employments, but still they can be harnessed with others to a cart or a roller, and thus kept from the indulgence. When they are not physically fit even for this mode of exercise their dress can be so arranged that their genitals are inaccessible to them, but this expedient is far less successful.

Faulty habits as they are called, that is to say, the emptying of the bladder and rectum into the clothes, are very frequent among the insane, and are due, sometimes to the mere negligence of dementia, sometimes to the desire to cause trouble and annoyance, and sometimes to a strange but undoubted association between the performance of these acts and the satisfaction of sexual desire. In most cases the fault can be overcome by observance of the routine of visiting the closet at moderately frequent intervals, that is to say, after every meal, and once or twice during the night.

Destructiveness is sometimes an impulsive outbreak of Berserker rage, of which no explanation can be given, and against which scarcely any precautions are adequate; but more often it is the resource of vacuity. A man has a certain amount of energy to expend, and this energy will find egress in some mode of activity. If no useful mode of activity presents itself or is available, it will find egress in twisting the buttons off his coat, in tearing

his clothes, in pulling things to pieces, and so forth. The remedy is obvious. He should be found something to do. If he is too demented to undertake any more intelligent occupation than that of tearing, he should be given newspapers to tear.

Mechanical restraint is practically never used in institutions specially devoted to the treatment of insanity, although the imbecile wards of workhouses are not considered properly equipped without a supply of strait waistcoats and of buckled sheets for fastening troublesome patients down in bed. While restraint to the extent which this provision of appliances implies is never required, and is improper, it is a question whether the total abolition of mechanical restraint is not an unnecessary and an undesirable sacrifice to convention and sentiment. Certainly mechanical restraint should never be employed, and should be rendered illegal outside of lunatic asylums; but with the usual logic of that law, which its professors are fond of describing as the perfection of common sense, the use of mechanical restraint, in the last resort, in places in which its use is surrounded by every imaginable safeguard, is pounced upon, discountenanced, and considered disreputable; while its use by persons who are very incompetent judges of its necessity or desirability as a frequent means of control in cases in which it is neither necessary nor desirable, is not forbidden in private houses or in workhouses.

While the cases in which the use of mechanical restraint is permissible are undoubtedly very few, there is a small residue in which it is sometimes of service and even very beneficial. A patient will sometimes beg to be tied down or put in a strait waistcoat in order to prevent him from giving way to his impulse to injure himself or others; and in such a case the feeling of confidence and peace that is engendered by the restraint is far greater than can be attained by the knowledge of watchfulness on the part of his attendants, and is well worth the sacrifice of a principle which, carried to excess, is but a sentiment. Cases of very persistent masturbation are sometimes impossible to control by any but mechanical means, and in such cases the mechanical restraint is certainly a less evil than continual struggling and interference. The same may be said of cases of very persistent self-injury. In this, as in other matters, there is a degree of fanaticism which is unjustifiable and pernicious. While it is undoubted that the use of mechanical restraint should be minimised to the utmost, it is a mistake to suppose that it would be an unmixed benefit to abolish it altogether.

Insects. *See* Cholera, Epidemic (*Etiology, Insects*); Dermatitis Traumatica et Venenata (*Causal Agents*); Malaria; Myiasis; Parasites (*Diptera*); Pinta (*Etiology*).

Insemination. — The deposition of semen in the vaginal canal as the result of sexual connection.

Insensibility. *See* Anæsthesia; Unconsciousness; etc.

Insertion of Cord. *See* Fœtus and Ovum, Development (*Umbilical Cord*); Pregnancy, Pathology of (*Diseases of Placenta and Cord, Anomalies in Insertion of Cord*).

Insolation. *See* Sunstroke; Tropics, Unclassed Fevers of.

Insomnia. *See* Sleep, Normal and Morbid; Brain, Affections of Blood-Vessels (*Anæmia, Hyperæmia*); Gout (*Symptoms, Nervous System*); Headache (*Results*); Heart, Affections of Myocardium and Endocardium (*Treatment, Sleeplessness*); Hypnotics; Hypnotism (*Therapeutic Uses*); Insanity; Pellagra (*Symptoms, Insomnia*); Pregnancy, Affections and Complications (*Nervous System*); Pruritus (*Treatment of Insomnia*).

Inspiration. *See* Physiology (*Respiration, Mechanism, Movements of Respiration*); Pulse (*Pulse and Respiration*); Respiration.

Instillation.—The administration of a liquid medicine drop by drop, usually in connection with diseases of the eye or ear. *See* Bladder, Diseases (*Cystitis, Treatment, Instillation*).

Instinct. *See* Insanity, Nature and Symptoms (*Index of Mental Functions, Instincts*).

Instruments. *See* Aseptic Treatment of Wounds; Antiseptic Surgery; Curettage; Disinfection; Labour, Operations; etc.

Insufficiency. *See* Inadequacy; Incompetence; Heart, Affections of Myocardium and Endocardium (*Valvular Lesions*); Pulse.

Insufflation.—The blowing of air laden with a finely divided drug or of a vapour into a cavity, *e.g.* the larynx for medical purposes. *See* Pharmacology; etc.

Insula Reilli. *See* Brain, Physiology of (*Anatomy, Island of Reil*).

Insular Sclerosis. *See* Paralysis (*With Tremor, Disseminated Sclerosis*).

Insurance. *See* Life Insurance.

Integument. *See* Skin.

Intelligence. *See* Insanity, Nature and Symptoms (*Index of Mental Functions, Thought, Intelligence*).

Intemperance. *See* Alcoholism; Morphinomania; Vice.

Intention. *See* Aseptic Treatment of Wounds (*Healing*).

Intention Tremor.—Trembling or oscillation of a part (*e.g.* the hand) when a voluntary movement is made; volitional tremor; a symptom of multiple or insular sclerosis. *See* Tremor.

Inter-.—In compound words *inter-* signifies *between*, e.g. *interarticular*, between the joints or within the joint between two bones in articulation; etc. etc.

Interception System.—The dry method for the removal of sewage; conservancy system; ashes, earth, peat, or sawdust may be used. *See* Sewage and Drainage.

Intermarriage. *See* Consanguinity.

Intermaxilla. *See* Palate (*Hare-Lip*).

Intermenstrual.—Occurring between two menstrual periods, *e.g.* intermenstrual pain (*Mittelschmerz*), intermenstrual discharge, red or white (*Metrorrhagia* and *Leucorrhœa*). *See* Gynæcology, Diagnosis in; Menstruation and its Disorders.

Interments. *See* Burial Places; Cremation.

Intermittent.—Occurring at intervals, *e.g.* intermittent fever (malaria), intermittent claudication (lameness due to arterio-sclerosis), intermittent pulse, intermittent insanity, etc.

Interne.—The physician or surgeon who resides in a hospital and is responsible for the patients within the building.

Interstitial.—Affecting the connective or indifferent tissue of a part or organ, as opposed to *parenchymatous*.

Intertrigo. *See* Erythema (*Due to Local Irritation*); Skin Diseases of the Tropics (*Caused by Climatic Conditions, Intertrigo*).

Intestines, Diseases of (Medical).

See also Intestines, Surgical Affections of; Abdomen, Injuries of; Abdominal Tumours, Diagnosis of; Actinomycosis (*Intestine*); Anæmia, Pernicious (*Treatment, Intestinal Antiseptics*); Anthrax; Breath (*Intestinal Irrigation in Uræmia*); Burns and Scalds (*Pathology, Intestinal Lesions*); Children, Development of (*Intestine*); Cholera, Epidemic; Cholera Nostras; Colon, Diseases of; Diarrhœa; Digestion and Metabolism; Dysentery; Enteroptosis; Fæces; Gall-Bladder and Bile-Ducts, Diseases of; Gastro-Intestinal Disorders of Infancy; Hæmatemesis; Heart, Affections of Myocardium and Endocardium (*Effects of Cardiac Disease, Passive Congestion of Intestine*); Heart, Affections of Myocardium and Endocardium (*Symptomatology, Intestines*); Hydatid Disease; Hysteria, Surgical Aspects (*Intestinal Obstruction*); Indigestion; Insanity, Nature and Symptoms (*Etiological Varieties*); Joints, Diseases of (*General Health in Tuberculous Joint Disease*); Lardaceous Degeneration (*Alimentary Canal*); Leucocytosis (*Lymphocytosis in Intestinal Catarrh*); Liver, Diseases of (*Chronic Venous Congestion*); Liver (*Tropical Abscess, Discharge into Intestine*); Liver (*Hydatids, Rupture into Intestine*); Lung, Tuberculosis of (*Complications, Alimentary System*); Medicine, Forensic (*Injuries, Ruptures of Internal Organs*); Melæna; Ovaries, Diseases of (*Complications, Intestinal Adhesions and Obstruction*); Nephritis (*Clinical Features, Diarrhœa*); Palpitations; Parasites; Pancreas, Diseases (*Pancreatitis, Acute, Diagnosis*); Physiology, Food and Digestion (*Structure, Intestinal Digestion*, etc.); Plague (*Intestinal Type*); Post-Mortem Methods (*Examination of the Body Cavities*); Spleen, Surgery of (*Movable Spleen, Diagnosis from Intestinal Obstruction*); Stomach and Duodenum, Diseases of; Stools; Tabes Dorsalis (*Symptomatology, Intestinal Crises*); Tuberculosis (*Alimentary Tract*); Typhoid Fever.

Anatomical and Physiological Considerations.—It is of importance in the study of diseases of the intestine to have a clear knowledge of the development of the intestinal tract. At an early stage of the human embryo, while the three layers of the epi-, meso-, and hypoblast still remain distinct, and the neural canal and notochord are in the process of formation, the

forward growth and over-folding of the mesoblastic layers immediately in front of the anterior end of the neural groove, form the head of the embryo in which the blind fore gut appears, while at a slightly later period a protrusion from the posterior blind end of the groove curves downwards and forwards to form the hind gut.

Coincident with the formation of the fore and hind gut, the lateral sulci, as they dip down, mark off the body cavity of the embryo from the rest of the blastoderm to form the middle part of the alimentary canal, which for a time remains open, and then gradually becomes pinched off from the yolk sac. Later, by invagination of the epiblast anteriorly and posteriorly, the mouth and anus are formed.

The further development of the pharynx, œsophagus, and stomach continue, while the intestine still retains a connection with the yolk sac by its duct. This gradually becomes constricted by ingrowth from the sides, remaining for some time as the omphalo-mesenteric or vitelline duct, the remains of which in after-life are represented by the permanent cicatrix known as the umbilicus. It thus happens that the whole of the intestine, with the exception of the invaginations of epiblastic tissue from without to form the mouth and pharynx and anus, is formed from hypoblast. The organs of secretion which discharge their contents into the intestine (liver, gall-bladder and its biliary ducts, pancreas, gastric and intestinal glands, etc.) are developed round tubular extensions from the intestinal tract, and are also hypoblastic structures, with their excretory ducts lined by epithelium of hypoblastic origin. The mesentery and peritoneum are developed from mesoblast. It is of interest in this connection to note that most primary malignant diseases of the intestine commence in the glandular structures, and are of hypoblastic origin and of a cancerous nature, and that primary tumours of the peritoneum are sarcomatous in structure.

Anatomically the intestine, about 26 feet in length, is divided into large and small, the former being one-fifth the length of the latter, but in diameter twice as great. The small intestine is arbitrarily divided into three portions, the first 10 or 12 inches, below the pylorus, being known as the duodenum, the upper two-fifths of the remainder the jejunum, and the lower three-fifths the ileum. The duodenum is, except when immediately adjacent to the pylorus, practically fixed, while the jejunum and ileum are freely movable by virtue of their being possessed of a mesentery. The large intestine, commencing at the lower end of the ileum, is divided into the cæcum, to which the rudimentary vermiform appendix is connected, the ascending, transverse and descending colon, the sigmoid flexure, and the rectum. The cæcum is that part of the large intestine which, blind at its lower end, lies below the level of the ileo-cæcal valve, and, unlike any other part of the intestine, is entirely covered by peritoneum. The sigmoid, the ascending and the descending colon have normally no mesentery, and are more or less fixed, but the sigmoid flexure, though usually situated in the left iliac region, is capable of extensive movement within the abdominal cavity, a fact of considerable clinical importance. The structure of the small intestine, speaking generally, is the same throughout its length. It has a serous coat, a muscular, a submucous and a mucous layer. The serous coat entirely surrounds the intestine, except at the mesenteric attachment. The duodenum is but partly surrounded, and the mesentery of the colon, and upper part of the rectum, contains numerous little projections enclosing fat, and known as appendices epiploicæ. The muscular coat is disposed in two layers, an external longitudinal and an internal circular. In the small intestine the disposition of the longitudinal coat is uniform, but in the large intestine it is so arranged as to constitute three well-marked longitudinal bands. The submucous coat is a layer of areolar tissue between the muscular and mucous coats. The mucous membrane is lined throughout with columnar epithelium, and contains the various secreting and absorbing structures necessary to digestion and absorption. Its superficial area is greatly increased by the presence, from immediately below the opening of the bile and pancreatic duct, of the valvulæ conniventes. These are very numerous, high in the jejunum, but fade gradually until they disappear towards the lower part of the ileum. The intestinal villi on and between the valvulæ conniventes are in structure similar to the mucous membrane itself. They are covered with columnar epithelium, and below this a basement membrane is found. In their structure also are included the lacteals, blood-vessels, and nerves, which are supported by a loose network of lymphoid tissue, with a few muscular fibres. The glands of the mucous membrane are Lieberkühn's, Brunner's, Peyer's patches, and solitary glands, which are known in the large intestine as lymphoid follicles. Brunner's glands occur in the duodenum only, and are branched tubular glands identical with the pyloric glands of the stomach. They lie in the submucous coat, and their ducts pierce the mucosa. Lieberkühn's glands are simply tubular or test-tube-like glands. They occur throughout the length of the intestine, but are more numerous in the large than in the small intestine. They are lined with a low, cylindrical epithelium between which are numerous goblet cells, these being more numerous in the glands of the large intestine. Lieberkühn's, and to a less extent Brunner's glands, secrete the succus entericus. The solitary glands are found throughout the large and small intestine, and in

all parts of their circumference. They are the size of millet seeds, and consist of lymphoid tissue. Peyer's patches are collections of the above lymphoid nodules arranged together in groups, of an oblong outline. They vary in length from one to three or four inches, are most numerous in the lower part of the ileum, are not found below the ileo-cæcal valve, and rarely occur as high as the upper part of the jejunum. They are situated opposite to the mesenteric attachment, and their long axis corresponds to the long axis of the intestine. These patches are best marked in youth, and after middle age begin to atrophy, and in old age are seen frequently only as pigmentary discolorations of the mucous membrane. Typhoid ulcers are the result of their destruction. The arterial blood-supply to the intestine is for the most part derived from the mesenteric arteries, but the duodenum is also supplied by several other branches, notably from the gastro-duodenal. The branches of the mesenteric artery run transversely below the serous coat, then perforate the muscular coat, and form a close network in the submucosa, which gives off branches freely to the vascular mucous membrane. Each villus is supplied by an artery which enters it excentrically. In the small intestine the veins accompany the arteries, but in the large intestine the arteries form a meshwork in the mucous membrane, and the veins return the blood in a manner similar to the blood-vessels of the mucous membrane of the stomach. Anatomically the anastomosis in the intestine is very free. In disease it would appear that under certain conditions the plugging of even a considerable branch by, say, a cancerous growth invading the vessel, can produce an annular gangrene of the intestine. The lymphatics or lacteals of the intestine commence as the lymph vessel or space in each villus. These unite with other vessels to freely provide the mucous membrane with a form of plexus in the submucous coat. Another plexus exists between the muscular coats, and these two uniting with the lymphatics under the serous coats on the mesenteric border form larger vessels, which empty themselves into the mesenteric glands, which again discharge their contents into the thoracic duct. The nerves of the intestine are derived from the solar plexus, and more particularly from the superior and inferior mesenteric plexus. The nerves accompany the arteries, perforate the muscular coat, and between the two layers form Auerbach's plexus, and in the submucous coat Meissner's plexus. Narrow filaments from Auerbach's plexus supply the muscular coat, and from Meissner's plexus filaments pass into the mucous membrane and the villi, but their ultimate termination is doubtful. The solar plexus from which the nerve supply of the intestine is derived is formed by the great semi-lunar ganglia which receive branches from the splanchnics and vagus. Stimulation of the vagus nerve increases peristalsis, and stimulation of the splanchnics inhibits movement if the blood in the intestinal vessels is normal, but if the blood in the intestinal capillaries is venous, the reverse effect occurs. The splanchnic is in addition the vaso-motor nerve of the intestine, and controls the largest vascular area in the body. It is also the afferent sensory nerve. Under Digestion and Metabolism the physiological functions of the various structures above enumerated in relation to secretion and digestion of fluids, and absorption of digestive products, are discussed. There the digestion of proteids, carbohydrates, fats, etc., by the various juices and their enzymes, with their subsequent absorption by the vessels, lacteals, etc., of the small and large intestine, is described. Intestinal digestion is most active in the small intestine, unless perhaps the greater activity of bacteria in the large diminishes the difference. Recent researches on absorption in the large intestine are of some importance clinically. Vaughan Harley's recent work on this subject deserves notice. Among other conclusions based on the experimental removal of the large intestine, the following occur:—

(*a*) Carbohydrates are absorbed equally well with or without the presence of the large intestine.

(*b*) Proteids are markedly influenced, 10 per cent less absorption occurring when the large intestine is removed.

(*c*) Fats are split up and absorbed equally well with or without the large intestine.

(*d*) The fæces are greatly increased in quantity (five times). This is principally due to the increased quantity of water, that is to say, a large quantity of water is absorbed in the large intestine.

(*e*) Urobilin is not discovered in the fæces after the large intestine is removed. Urobilin is the product of the splitting up of the bile pigments in the large intestine by putrefactive action, probably chiefly bacterial. Under normal conditions urobilin exists in the large intestine, the fæces, and the urine, and is recognised by giving a deep pink reaction with a concentrated solution of perchloride of mercury. Excessive increase in the urine and fæces indicates either an abnormal secretion of bile, or increased putrefactive action in the intestine. Thus we expect to find in diarrhœa of a septic nature an increase of this substance in the urine.

Bacteria of the Intestinal Tract

Before birth the intestinal tract is sterile. The meconium for three to seven hours is free from bacteria, or at the most a few cocci are obtainable. In a few hours bacteria make their appearance in abundance, and throughout life the intestinal tract from mouth to anus abounds

in innumerable varieties. It may be said at once that attempts to isolate and classify the bacteria found in the intestine are most incomplete, for the task is herculean.

The Rôle of Bacteria in Health.—Bacteria or spores are introduced into the stomach at every act of deglutition. Many are destroyed, some are aerobic, and by being deprived of oxygen, die; others are destroyed by the hydrochloric acid of the stomach, which is a germicide, while others find no suitable pabulum for their nutrition and growth. By the germicidal action of the hydrochloric acid of the gastric juice the formation of decomposing products and other disturbances produced in the normal course of digestion is prevented. The comma bacillus of cholera is readily destroyed by the gastric juice, as is also the bacillus of splenic fever, though its spores are unaffected. Many pathogenic bacteria are unharmed by the gastric juice, *e.g.* tubercle bacilli. It is a noteworthy fact that the average amount of hydrochloric acid found in the gastric juice practically coincides with the amount required to kill experimentally most fermentative and many pathogenic bacteria.

The Action of Bacteria on Digestion.—In the normal state of the intestinal tract bacteria aid the various secretions and their enzymes, but to what extent is doubtful. Growth and normal nutrition can go on without the presence or assistance of bacteria, as has been proved experimentally by placing newly-born animals in sterile media and feeding them on sterilised food, when they grow and thrive as satisfactorily as under their usual conditions. In the stomach, on account of the action of hydrochloric acid, little digestion by bacterial action takes place. In the early stages of gastric digestion, before the hydrochloric acid is fully active, carbohydrates undergo, to a slight extent, lactic acid fermentation, but proteids and fats are not attacked to any extent, if at all. In the intestine the bacterial action is more vigorous. The less acid reaction of the intestinal contents favours this, but it is also supposed that organic acids are set free by the action of bacteria on carbohydrates, and that these acids to some extent inhibit bacterial action on the putrefaction of proteids. A condition favourable to their activity and one antagonistic appear therefore to go on side by side. Proteids in the intestine are first slowly dissolved and converted into albuminoses and peptone; then these along with the tyrosin of tryptic digestion are broken up into various aromatic compounds, such as indol, skatol, and skato-carbonic acid by bacterial action. Carbohydrates are affected earlier than proteids. Starch is practically decomposed into sugar, and sugar already converted is further decomposed into lactic, butyric, and succinic acids. What change is affected on cellulose during digestion is entirely by bacterial action, or, otherwise stated, bacteria are the only agents which alter cellulose. On fats under normal conditions little action is produced by bacterial action, but in the absence of bile or pancreatic juice they are converted into fatty acids, which pass out of the body unabsorbed in the fæces.

The Rôle of Bacteria in Disease.—Pathogenic bacteria may be introduced into the intestine and directly cause specific disease (typhoid, tubercle, etc.), or those already existing in the intestine may, under altered conditions of nutrition, multiply and be converted from saprophytic into pathogenic organisms. The exact conditions under which saprophytic bacteria become pathogenic are very imperfectly understood, but it would seem as if a simple abrasion of the mucous membrane, the presence of enteritis, defective circulation, local or more general, and even a lowered condition of the body, is sufficient to allow of this change. As illustrations we may mention that in some cases of appendicitis the inflammation is produced by the entrance of the bacillus coli into the mucous membrane eroded by an enterolith or other cause; or cases of uncomplicated enteritis from a mechanical irritant are followed by a putrid diarrhœa of bacterial origin. The intestinal wall in its healthy state effectively resists the passage of bacteria through it, but in certain diseased conditions, as, for example, gangrene following strangulated hernia, peritonitis, etc., bacteria are able to pass through the walls, and cause definite lesions in the intestinal wall itself and in the adjacent peritoneum. The pathological changes following may result either by direct action of the bacteria themselves or by their toxins.

In the intestinal digestion of proteids the putrefactive bacilli which produce poisonous nitrogenous and alkaloid bases known as ptomaines do not flourish under normal conditions, and consequently none are normally present in the intestine. They may be, however, introduced through the medium of tainted meat, either as bacteria capable of elaborating these bases, or as ptomaines already elaborated.

Authorities are not in agreement as to the period of production of ptomaines in the process of putrefaction, some holding that they are late, others that they are early products. If a single variety of intestinal bacterium becomes abnormally active, it is probable that others, on account of the altered conditions produced, will become pathogenic also.

A few of the more important bacteria found in the intestine deserve special attention. 1. Bacterium coli communis is present in both the small and large intestine in health, and to it are ascribed many of the useful physiological functions of intestinal bacteria. Under normal conditions it is saprophytic, but under altered conditions of nutrition of the intestine or

perhaps of the body generally, it becomes pathogenic and plays an important part in the causation if it is not the sole cause of many intestinal diseases. In most inflammatory and suppurative conditions of the intestine it is found in excess. In suppuration of the gall-bladder and bile ducts it has a most important place, also in some varieties of enteritis, in appendicitis, and in peritonitis, whether the intestine is perforated or not, as it can pass through a damaged intestinal wall and produce a distinct variety of peritonitis. In typhoid fever the colon bacillus increases enormously, especially in the large intestine, and its resemblance to the typhoid bacillus requires special cultivations on various media and discriminating reactions to distinguish one from the other.

2. Staphylococci are found in small numbers in the normal intestine, but in catarrh of the mucous surface, and in suppurative and gangrenous conditions and other diseases, they multiply vigorously.

3. Streptococci, which normally exist in the bowel, are virulent in diseased conditions and abound in all inflammations with fibrous exudations, *e.g.* non-diphtheric inflammations, enteritis, and colitis, etc. It is stated that streptococcus longus in the milk of cows suffering from mastitis produces diarrhœa in children, and when experimentally injected into animals.

4. *Bacillus Enteritis Sporagenes.*—Klein isolated this anaerobic bacillus in epidemic diarrhœa, of which milk was supposed to be the cause. His own and further investigations by others have not confirmed this view, but rather go to show that the bacillus exists widely in nature, in the normal intestine, in normal dejecta, in dysentery, diarrhœic stools, ulcerative colitis, etc. Gautner in 1888 isolated a bacillus which he calls bacillus enteritis in some cases of meat poisoning, and another bacillus evidently of the same group has lately been isolated in cases of meat poisoning. Both caused intense hæmorrhagic enteritis.

5. Bacillus pyocyaneus is said to cause gastro-intestinal catarrh, and to be found in the intestine in cases of infantile atrophy.

6. The bacilli of typhoid fever are found chiefly in Peyer's patches and the solitary glands before the stage of necrosis and suppuration, when they to a great extent disappear, remaining at the edge of the typhoid ulcer and in the lymphatic vessels and the mesenteric glands. It is probable that the necrosis is chiefly brought about by the action of the toxic products of this bacillus. As already mentioned the colon bacillus is found in great abundance in combination with the typhoid bacillus.

7. Bacillus of diphtheria is found whenever a true pathogenic membrane forms in the intestine, and where the diphtheric membrane from the fauces is swallowed. It is by no means the only bacterium present in diphtheric membranes, but is associated with streptococci chiefly.

8. Cholerus spirillum (*vide* "Cholera," vol. ii. p. 115).

9. Tubercle bacilli may be present in the intestine and fæces in the absence of tuberculosis, yet their presence in the fæces in the absence of lung disease is most suggestive, especially in suspected cases of primary tuberculosis of the intestine in children (*vide* "Tuberculosis and Intestinal Tubercular Ulceration").

10. *Actinomycosis.*—For a description of the actinomycoses or ray fungus in the intestine *vide* "Actinomycosis and Appendicitis."

Malformations

1. *Of the Duodenum.*—Occur as duodenal pouches or occlusion.

Duodenal occlusion is met with just above the entrance of the bile duct into the duodenum. It may occur as a complete occlusion, the first portion narrowing gradually so as to present a blind end ununited to the second portion, except, possibly, by a fibrous band, or as a diaphragm complete or perforated, and consisting of structures similar to those composing the intestinal walls. A suggested method of production of both of these conditions is that a kink is produced above the bud when the hepatic diverticulum buds off from the duodenum, and adhesion produces permanent occlusion. The symptoms produced are vomiting, which occurs soon after birth, and the vomited matter is not bile-stained. Jaundice is usually absent, muconeum is passed, and the bowels may be moved several times after birth. Rapid emaciation follows and death takes place in from one to eleven days, the time varying with the degree of obstruction.

Duodenal diverticula or pouches occur in the same situation as occlusion, namely, just above the biliary papilla. The pouches are wide-mouthed, and their walls consist of the same structures as the intestinal walls. As a rule they do not attain to any great size, and are not known to give rise to any symptoms unless, as has been occasionally reported, gallstones or other foreign bodies become arrested in them. These diverticula appear to be connected with the hepatic offshoot in much the same way as Meckel's diverticulum is with the vitelline duct. They appear to be related to an erratic form of developmental activity at this portion of the intestine. The duodenum has also been found unattached to or occluded near the pylorus at its upper end, and a similar defect has been noticed near the jejunum at its lower end.

2. *Of the Small Intestine.*—(*a*) *Meckel's diverticulum.*—In early fœtal life the intestine communicates with the yolk sac by the vitelline canal, and a persistence of this communication may leave a tubular connection between the

umbilicus and the intestine. This may be occluded at any part of its course, and this remnant of fœtal life (met with in about 2 per cent of bodies examined) is known as Meckel's diverticulum. If the tube remains open at the umbilicus, it forms one variety of fæcal fistula (*vide* "Intestinal Obstruction").

(*b*) *Multiple diverticula* of the small intestine are said to have their origin in protrusions from within, the result of defective resistance of limited areas of the intestinal muscular coat, or dragging from without, the result of adhesions due to congenital peritonitis.

(*c*) *Occlusion of the small intestine* by diaphragms stretching across the lumen occurs singly as a rule, but is occasionally multiple. Their etiology has received no satisfactory explanation, but it has been suggested that they resemble the similar malformations in the duodenum, and arise from excess of the atrophic process which results in the obliteration of the vitelline duct. It is also possible that intra-uterine peritonitis may, by the formation of bands, produce the same result. Occlusion of the small intestine is incompatible with more than a few days of life, but as distinguishing symptoms from duodenal occlusion vomiting of biliary matter and abdominal distension will usually be recognised, the latter in proportion to the distance of the obstruction from the stomach (*see also* "Gastro-Intestinal Disorders of Infancy," vol. iii. p. 400).

(For malformations of the colon *vide* "Colon," vol. ii. p. 185.)

Enteritis

Definition.—In its widest sense enteritis implies inflammation of any part of the intestine large or small. This generic definition is, however, by common usage, restricted to inflammation of the small intestine. The corresponding inflammation of the large intestine is known as colitis, and is described under that heading.

Etiology.—Enteritis is caused by contact of an irritant of sufficient virulence with the intestinal wall. It may also be produced by an extension of inflammation from other neighbouring structures or organs, and it is then known as secondary enteritis.

Predisposing Causes.—(*a*) *Debility.*—Feeble and reduced health, particularly in very old or very young people, predisposes to enteritis.

(*b*) *Age.*—Children suffer far more frequently than do adults. Indeed a mild catarrhal enteritis is such a frequent accompaniment of the period of lactation as to make it probable that the highly active digestive tract of early childhood is specially susceptible to inflammation.

(*c*) *Season and Temperature.* — Atmospheric conditions and environment exercise a marked effect upon the prevalence of epidemic enteritis commonly known as summer diarrhœa. The third quarter of the year, July, August, and September, has double the number of deaths from this cause that the combined remaining nine months of the year have. In a dry, hot summer and autumn more cases of epidemic enteritis occur than in a cool wet season. It is more prevalent in the ill-ventilated and dirty streets of large towns than in more healthy localities, or in the country. The facilities for its spread are greater in crowded localities, but dirt, the want of sunshine, and fresh air among the children, and other circumstances favourable to bacterial growth, errors of feeding, spoiled food, and milk, etc., are all powerful factors in favour of its occurrence.

(*d*) *Exposure to cold* has the same relation to enteritis as it has to pleurisy or nephritis, but another factor, namely, disordered digestion by producing an intestinal irritation, may act as a more direct exciting cause.

Exciting Causes.—These may be mechanical, chemical, or bacterial. Of mechanical causes the most potent are indigestible and undigested food. For instance, the firm coagulum of milk may cause diarrhœa in children, unripe fruit too freely eaten, fruit containing much woody fibre, nuts, fruit-stones, gallstones, intestinal worms, etc. Among chemical irritants capable of causing enteritis may be mentioned violent purgatives such as croton oil; other irritants include the caustic alkalies, carbolic acid, arsenic, corrosive sublimate, etc. Under this heading also may be placed the unascertained irritant which causes enteritis in some cases of extensive superficial burns.

Bacterial causes of enteritis assume a more and more important place as our knowledge increases. The exact method of action of the micro-organisms is as yet imperfectly understood, but it is now generally recognised that the bacteria normally existing in the intestine may, under certain altered states of nutrition of the individual, of the intestine itself, or of its contents, become pathogenic, or that one species of bacterium, becoming abnormally active, may so alter the normal equilibrium that other species can multiply and become virulent; or bacteria introduced into the body and coming in contact with the intestine may cause inflammation.

In the former class probably the best example is furnished by the bacterium coli communis, which, normally saprophytic and useful under healthy conditions, may become under abnormal conditions pathogenic, as, for example, above the site of a strangulation, when the resulting peritonitis is due to this organism becoming unusually virulent and invading the intestine after its vitality and nutrition have been lowered by strangulation.

In the second class, bacteria introduced into the stomach and intestine from without, the medium is almost always some form of foodstuff which is more or less decomposed, as, for example,

tinned, preserved, and potted meat, tinned fish, putrefying meat, pork more frequently than other kinds, sour milk, high game, oysters, etc. In this class of food poisoning the poison may be either bacterial or the product of their action, which results in the formation of ptomaines, and the symptoms produced are known as ptomaine-poisoning (*vide* "Food," vol. iii. p. 334). There is every reason to believe that this is due to a poisonous animal alkaloid formed, it is generally supposed, in the later stages of putrefaction and not to bacteria themselves. The exact alkaloid has not yet been isolated, but a substance called "sepsin," capable of producing intense hæmorrhagic enteritis, has been obtained by Bergmann from decomposing yeast, and Briegar has secured nuerine, coaline, and murodine, which closely resembles the poison of mushrooms—muscarine—from decomposing meat. Alkaloids, resembling curara in their action, have also been separated from decomposing fish. All of the alkaloids thus separated are not poisonous, but some are extremely toxic. All toxic ones cause enteritis, cardiac failure, and paralysis or convulsions.

In epidemic enteritis (summer diarrhœa of children) it is probable that no one bacterium but many are the potent factors in its causation. Attempts have been made to classify cases according to the bacteria found in the stools, but such a classification is, for the present at least, impracticable. Klein separated from the stools in an outbreak of enteritis a bacillus which he called bacillus enteriditis sporagenes, but he and others, by subsequent investigations, have failed to prove this anaerobic organism to be the specific bacterium of this disease.

A break in continuity of the intestinal mucous membrane favours the entrance of bacterium coli, and enteritis, with subsequent ulceration, is likely to supervene, as exemplified in some cases of traumatic appendicitis, or intestinal cancer or intestinal obstruction when the intestine at and above the lesion is frequently inflamed and ulcerated, and may be gangrenous as a result of bacterial infection of the damaged intestinal walls (*vide post* "Bacteria of the Intestine").

Pathological Anatomy.—The superficial mucous membrane is in mild cases the site of inflammation, but in more severe forms inflammation extends to the submucous coat. The initial lesion appears as a diffuse redness of the mucous membrane, and the exudation of inflammatory products causes œdema and tumefaction. The surface of the inflamed mucous membrane is usually coated with a tenacious layer of mucus, often blood-stained. Lieberkühn's follicles, Peyer's patches, and the solitary glands are swollen and prominent; their ducts become obliterated, small erosions or catarrhal ulcers develop, and, if the inflammation is severe, hæmorrhage into and under the mucous membrane takes place. Unless the case becomes chronic or the inflammation be very acute, the muscular and serous coats are slightly, if at all, affected. If, however, the case becomes chronic, the inflammatory products develop into new connective tissue, the glandular elements are to a great extent destroyed, the muscular and serous coats become involved, the intestinal wall is infiltrated by the products of chronic inflammation, the mucous membrane at the same time shows considerable erosion, and by sloughing of the glandular elements, consequent on tension produced by plugging of their ducts, catarrhal ulcers are formed. The secretive and muscular functions become then permanently damaged. In certain cases further mischief may follow; localised abscesses may form and break into the bowel, or gangrene, with perforations of the bowel, may result.

Enteritis rarely affects any considerable length of the small intestine; usually a few yards only are involved. The colon is more frequently affected than other regions, and the jejunum probably less frequently than any other part.

Symptoms.—The symptoms of acute intestinal catarrh vary greatly in severity, and to a large extent in accordance with the cause. An acute irritant, like carbolic acid or a caustic alkali, will produce a more acute diarrhœa than that due to a simple mechanical irritant. As a rule, the earliest symptom, as in most febrile affections, is chilliness. A feeling of uneasiness in the abdomen, followed by colicky pains, nausea, and loss of appetite, are complained of early. The nausea may be followed by vomiting and hiccough. The tongue is dry and coated, the expression is anxious, and the face pinched; thirst is complained of, diarrhœa soon commences, the stools being at first semi-solid and gruel-like, but later watery, acid, and scalding, containing more or less mucus in accordance with the site of the enteritis; the higher in the intestine the less the mucus in the stools. (A stool almost wholly composed of mucus may with a fair degree of certainty be said to have its origin below the splenic flexure of the colon, *vide* "Fæces," vol. iii. p. 241). The stools are usually numerous, and often become fœtid, either from decomposition in the intestine by bacterial action or from temporary arrest of the biliary secretion. The colour may be green from biliverdin staining, especially in children, but as a rule the evacuations remain clear and watery, consisting of unabsorbed intestinal contents—epithelium, mucus, food particles, leucocytes, other products of inflammation, and innumerable bacteria, of which the bacterium coli spores and rod-shaped bacteria are the chief. In proportion to the amount of fermentation going on in the intestine there will be abdominal distension, borborygmi and increased flatus passed per anum, and palpitation and cardiac discomfort may arise from upward displacement of the diaphragm. Diarrhœa may be absent in inflammation of the duodenum or of the upper part of

the jejunum, the remaining healthy intestine below absorbing the surplus fluid before it reaches the rectum. In children and old persons more particularly emaciation may be rapid, and strength quickly lost, especially if the diarrhœa be profuse. Urine is scanty and high-coloured. The temperature is variable, but as a rule is not high. Pulmonary œdema and cardiac failure in severe cases complicate the disease, and occasionally epileptiform convulsions supervene towards the end. In accordance with the cause of the enteritis the symptoms necessarily vary.

The symptoms of ptomaine-poisoning call for special notice. At a variable interval, an hour or two, or as long as thirty-six hours, after eating tinned salmon, high game, etc., the patient complains of a feeling of nausea, restlessness, and sickness. This is speedily followed by vomiting and gradually increasing abdominal pain, which soon becomes agonising. Muscular cramps and clonic spasms, especially in the legs, add to the patient's misery, and soon a feeling of intense prostration, accompanied by cold sweats, nervous depression, and cardiac failure, appears. The expression is anxious and even terrified; diarrhœa usually supervenes early, is profuse, watery, often fœtid, and sometimes bloody. Intense thirst is nearly always complained of, the pulse is quick and small, the urine is scanty, and the temperature varies from 100° to 103°. An urticarial or erythematous rash is not uncommon. If the dose of the poison has been large, and especially if diarrhœa does not early carry off some of the poison, the patient may lapse into a state of unconsciousness and coma. The pulse becomes quicker and more feeble, and cardiac inhibition becomes gradually more marked before death.

The severity of the symptoms varies not only with the amount of the poisonous dose taken or elaborated after ingestion, but different individuals who have partaken of the same meal and in similar quantities are not equally affected. Assuming that the poison is equally distributed in the food, personal idiosyncrasy must enter into the calculation; but there are many fallacies.

The symptoms begin to abate at a variable period of a few hours or days, but in any case convalescence is slow.

Epidemic enteritis of children, otherwise called summer diarrhœa, or septic diarrhœa, is characterised by the sudden onset of vomiting and diarrhœa. The stools—at first frequent, offensive, and solid—later consist of watery serum, mixed with flaky mucus. This disease is further characterised, on account of the rapid loss of fluid through diarrhœa and vomiting, by rapid emaciation, intense thirst, and collapse, which may in a single day result in death (*vide* "Gastro-Intestinal Diseases of Infancy and Cholera Nostras").

Diagnosis.—A history of improper feeding, followed by colic, nausea, diarrhœa, rise of temperature, and quickened pulse, indicates the nature of the case. It is, however, often very difficult to determine the part of the bowel affected. Catarrh of the duodenum may be accompanied by jaundice, and often is by gastric catarrh; or pain localised in the right hypochondriac region may suggest the site. The diagnosis of inflammation of the jejunum from that of the ileum is generally impossible. The distinctive points between inflammation of the small and large intestine are not very definite; but if the stools are greyish-yellow and not numerous, and are mixed with partially digested food, and if colicky pains are severe, and if the urine shows a burgundy-red colour, on the addition of nitric acid, and this persists after boiling, indicating the presence of indican, the chances are in favour of the small intestine being the site of the lesion.

Care should always be exercised in deciding whether any given case of enteritis is primary or secondary; that is to say, whether it is the only and primary lesion, or whether it is but a symptom of another disease (*vide infra*, "Chronic and Secondary Enteritis").

Prognosis.—Prognosis is as a rule favourable. The majority of cases recover in a few days after the cause has been removed. In the case of ptomaine-poisoning recovery as a rule occurs, though many cases are serious and demand investigation by the local authorities. Epidemic enteritis of children should always be regarded seriously, for it is remarkable that children apparently but slightly ill in the course of a day or two lose ground so rapidly that they die before one has almost time to realise that any serious danger exists.

Treatment.—The most important matter in the treatment of all cases of intestinal catarrh is to ascertain its cause and so determine whether the remedy should be directed to the removal of an irritant or to subduing the irritability of the inflamed mucous membrane. Slight cases practically cure themselves; the diarrhœa being salutary removes the irritant naturally. After this, restriction in diet or abstention from food temporarily, and perhaps a small dose of opium (pulv. ipecac. co. grs. v. in an adult) may be all that is required. If the cause is an irritant, such as undigested food, a good treatment is to administer a dose of castor oil with 8 to 15 mms. of tr. of opium, and to follow this by a further dose of opium (pulv. ipecac. co. grs. v., and repeated as necessary) to allay peristalsis and give the intestines rest. Having removed the irritant, if the symptoms still persist on account of resulting inflammation, or having determined that the case is one of enteritis not caused by an irritant lying in the bowel, recourse must be had to a combination of sedatives and astringents. Mist. cretæ of the B.P.

finds most favour, and may be given alone or in combination with opium, as the mistura cretæ cum opio with kino or catechu added. Chalk acts by saponifying the acid fats, and thus removes acid irritants, at the same time coating the inflamed mucous membrane with a protective and soothing covering in a similar manner to the heavy insoluble bismuth salts (bismuthi trisnit. grs. x., pulv. ipecac. co. grs. v. 4 ter hor.) which are also very useful remedies. If colicky pains are severe, nothing gives such speedy and satisfactory relief as a hypodermic injection of morphia, and with this may be combined the application of warmth in the form of fomentations covered by a waterproof, or a hot poultice over flannel on the abdomen, either to be changed frequently.

If the case under consideration is one of diarrhœa from eating spoilt food or decomposing meat, a dose of castor oil (℥j.) should be administered; but if any of the irritating meal remains in the stomach, it should first be ejected by an emetic. Agonising colic and muscular cramp demand a hypodermic injection of morphia, and a stimulant is required to combat the reflex cardiac failure and the depressing action of the ptomaine on the nervous system. In the case of mushroom-poisoning the antidote to the active principle, muscarine, is belladonna or atropine, and this should be given in 10 or 15 mm. doses of the tinct. belladonnæ, or one-hundredth of a grain of atropine every hour until dryness of the mouth and pupillary dilatation are detected. In enteritis, the result of arsenical poisoning, if there is reason to believe that the arsenic has not already been absorbed, or in other words if the case is seen early, ferric hydrate should at once be freely given; but if the case is seen late, the enteritis resulting can only be treated on general principles. Ferric hydrate can be readily prepared by the addition of liquor ammon. fort. to either the tr. or liquor ferri perchlor., care being taken that excess of ammonia is not present before drinking.

The important part played by micro-organisms in many cases of enteritis has given rise to a useful method of treatment which consists, after the initial administration of a purgative, in the administration of one or other of a class of drugs known as intestinal antiseptics, and of frequent alteration in the diet. By means of the drug we hope to inhibit the growth of the pathogenic bacteria, and by alteration of the diet to starve the bacteria by depriving them of the pabulum on which they feed and multiply. The most useful intestinal antiseptics are the preparations of mercury, in the form of either liquor hydrarg. perchlor., hydrarg. cum cretæ, or calomel. Of these, liquor hydrarg. perchlor. is by many regarded as the best. Calomel, in spite of evidence that it diminishes bacterial putrefaction in the intestine, as shown by the increased amount of bile pigment in the fæces, which has not been converted into urobilin in the large intestine, and by the diminution of aromatic sulphates in the urine, has the drawback that it acts as a purgative partly by increasing the flow of bile, and also by being an irritant, but it is often apparently useful. The salicylic acid group is excellent, and probably salol is preferable to salicylic acid or beta naphthol or salicylate of bismuth. Mercury may be advantageously combined with bismuth and soda, as in the following for an adult:—

℞ Liquor hydrarg. perchlor. ♏xx.; sodæ bicarb. bismuthi subnit. āā, grs. v.; mucilaginis, q.s.; aqua anethi, ℥ss. Ft. mist.

Or, if salol is preferred, it may be given to an adult in doses of 10 to 15 grains, alone, or with carbonate or salicylate of bismuth. Salol has this great advantage in enteritis, that it passes through the stomach unchanged and breaks up in the intestine into salicylic acid and phenol. Resorcin in doses of 2 to 5 grs. is sometimes used in combination with salicylate of bismuth.

With regard to the second part of this method of treatment, viz. frequent alteration of diet, if the child is bottle-fed, ordinary milk should at once be stopped and recourse had to sterile milk. If this is unsatisfactory, milk should be replaced by animal broths, such as beef or veal extracts prepared at home, or given in the form of Liebig's extract, or similar commercial preparations, with a carbohydrate, *e.g.* ground rice. A very useful form of beef tea is prepared by the cold method. This is prepared by adding 10 drops of dilute hydrochloric acid to half a pound of finely minced beef and half a pint of cold water, which are allowed to stand for an hour and are then strained through muslin. The desired quantity is then placed in a cup and warmed (not boiled) until the colour changes to the brown of ordinary beef tea. These alternations of diet should, in the event of the continuation of symptoms, not last longer than twenty-four hours, when further changes should be made to milk again, milk whey mixed with white of egg and cream, etc.

Chronic or Secondary Catarrh—Etiology.—(1) Acute primary catarrh may become chronic, especially in old and feeble subjects; (2) extension of inflammation from structures adjacent to the intestine; (3) from tuberculous or chronic ulceration of the gut (*vide* "Ulcers of the Intestine"); (4) the result of portal obstruction, hepatic or cardiac; and (5) in pyæmia, etc.

Morbid Anatomy.—The intestinal mucous membrane becomes thickened, fibrous, eroded, and studded with ulcers. The blood-vessels are congested; the muscular coat is generally, to some extent at least, hypertrophied, and later is replaced by fibrous tissue. The glands are destroyed, their ducts being in the first place blocked, and retention cysts form before their

final destruction. The villi, as a rule, are shrunken and atrophied.

Symptoms.—The acute form merges into the chronic gradually; the bodily health depreciates; the complexion becomes muddy; there is persistent diarrhœa; appetite is impaired; nausea is present; anæmia and loss of weight follow, and absorption of poisons from the intestinal canal cause listlessness and hypochondria. Constipation alternates with diarrhœa. This form of enteritis is more usually located in the large than in the small intestine. Prognosis is bad, for this is a secondary disease, occurring in the later stages of Bright's disease, heart disease, chronic pyæmia, etc.

Treatment is palliative; and the astringent and sedative remedies used in acute intestinal catarrh are employed. A bland, easily digested food, which yields but little residue, is desirable. Tonics, such as quinine and iron, if there is much anæmia, are useful. Change of scene with hydropathic treatment are very useful. Each case of chronic enteritis must be treated more on the principle of removal or amelioration of its original causative disease than by the administration of remedies acting on the intestinal tract.

Ulcers of the Intestine

(1) *Tuberculous Ulcers of the Intestine.*—Tuberculous infection of the intestine occurs as a primary or as a secondary disease. As a primary disease it is most frequently found in children, though it occurs in adults, in either case the tubercle bacilli being introduced through the medium of tuberculous milk, etc. As a secondary disease the most common mode of infection is by swallowing of phthisical sputum. It is, however, evident that any medium containing tubercle bacilli when swallowed may be the starting-point of tuberculous ulceration of the bowels. The intestine may also be the site of a miliary tubercle deposited there in a ease of general tuberculosis. It by no means follows that an individual who has swallowed one or many doses of tubercle bacilli gets intestinal tubercle. The bacilli must find conditions favourable to its growth, and in an individual predisposed.

Certain pre-existing lesions of the intestine appear to favour the deposition and growth of the tubercle bacillus, *e.g.* follicular ulcers, chronic non-inflammatory catarrh, etc.

Infeetion may also occur by extension from the peritoneum or by the discharge of tuberculous pus into the bowel.

Situation.—Tuberculous ulceration of the intestine is least rare in the cæcum and lower part of the ileum, and diminishes in frequency above and below this point, the duodenum being but rarely affected. The rarity of tuberculous lesions in the higher part of the intestine is attributed to the destructive or inhibitory effect of the acid of the stomach and upper part of the bowel on the tubercle bacillus.

Anatomical Characteristics. — Tuberculosis arising in the mucosa appears first as a number of little, hard nodular projections in the lymph follicles. The tips of the nodules then become grey and caseate, break down and separate, forming a number of small ragged ulcers. If the caseating nodules lie close together, they coalesce, or if a Peyer's patch is the site an ulcer is formed having the following characteristics:—The floor, formed of the submucosa, is rough, infiltrated, inflamed, and presents caseous nodules. The edges are sinuous and irregular, and slightly undermined. Tuberculous deposit and infiltration extend beyond the edge of the ulcer. The mucous membrane beyond the ulcer is for a certain distance inflamed, but if the ulcers are not close together healthy mucous membrane may intervene. The direction of the ulcer is generally transverse to the long axis of the intestine, and it is in shape oblong, but it may be round. Looked at from the outside of the bowel, the floor of the ulcer is seen to be thickened, and frequently small stellate cicatrices are visible, showing that the serous and muscular coats are involved. Adhesions may occur to the neighbouring coils from spreading inflammation, and perforation seldom occurs. Tuberculous are distinguished from typhoid ulcers by their transverse direction, by their irregular, ragged, and infiltrated edges, by their thickened bases, and by the existence of small caseous nodules in their proximity. Tuberculous disease begins in the lymph follicles, spreads by the lymphatics and infects the mesenteric lymph glands. The ultimate result of tuberculous ulceration is, in the vast majority of cases, death, because it usually occurs in the excavating stages of phthisis. Fifty per cent of phthisical patients have ulcers of the intestine. They may, though rarely, perforate, or may heal and cause stricture of the intestine at one or many points. More rarely healing probably takes place without their existence being suspected, and without leaving noticeable permanent damage to the intestine during life.

Symptoms.—If persistent diarrhœa occurs in phthisis, tuberculous ulceration of the intestine is the likely cause. Tuberculous ulceration, however, does not always produce diarrhœa, and as in non-tuberculous inflammation of the intestine, speaking generally, diarrhœa is more likely to occur the nearer the ulceration is to the rectum. Tuberculous ulceration of the large intestine nearly always gives rise to diarrhœa. Blood may be present in the motions, which are frequently fœtid on account of the bacterial putrefaction, and for the same reason tympanitis is often marked. Tubercle bacilli can, as a rule, be detected in the stools in cases of tuberculous ulceration of the intestine, but their absence is not positive proof of the absence of tuberculous

disease. In children, the subjects of primary tuberculous ulceration of the intestine, diarrhœa attended by loss of weight and strength out of proportion to its severity, loss of appetite and sickness, with abdominal tenderness and distension, and the existence of palpable glands in the abdomen, may make a diagnosis possible; but in the absence, after the lapse of some time, of other evidence of tuberculous affection, it is often impossible to have a positive opinion.

Diagnosis.—Lardaceous disease of the intestine may give rise to diarrhœa and recourse for diagnostic purposes may be had to the examination of the fæces for tubercle bacilli, and if these are regularly found in the stools, a diagnosis of tuberculous ulceration is justifiable. As lardaceous disease frequently coexists with phthisis, it is not unlikely that diarrhœa occurring in the last stage may be caused both by tuberculous and lardaceous disease.

Treatment.—The majority of cases occur in connection with pulmonary tuberculosis, and the intestinal lesion is both late and secondary. Remedies to alleviate the diarrhœa are the most generally applicable. The primary disease requires first attention, and in most cases this treatment consists in restraining the diarrhœa, diminishing the fermentation in the intestine, and regulating the diet, so as to avoid as much as possible exciting fresh inflammation in the highly susceptible intestine. For the diarrhœa, astringents are always useful, and of these chalk and opium, kino and catechu, give the best results. Opium by the mouth occasionally causes vomiting, and may then be tried as a suppository or an enema. Salol, in five or ten grain doses, is sometimes useful, as it restrains the diarrhœa and diminishes putrefactive fermentation and flatulence. It is said to inhibit the action of the putrefactive as well as the tubercle bacilli.

Attention to diet as laid down under "Enteritis" is useful, and it is well to give sterile foods, avoiding every article of diet calculated to irritate the intestine. By these means we may hope not only to restrain diarrhœa but to promote the healing of ulcers, and by avoiding fermentation to diminish abdominal distension and the risk of perforation. If much abdominal discomfort exists, warmth, applied to the abdomen as a poultice fomentation, or dry heat, is comforting. If general peritonitis exists, opium should be given freely if the case is hopeless, but it is possible that an occasional case of perforation from a tuberculous ulcer might be treated by adominal section, with the object of cleansing the peritoneum, and either of stitching the perforation or resecting the bowel at the site of the lesion. In children with primary ulceration of the intestine, the source of affection should be ascertained if possible, and perhaps fresh invasions be avoided by the substitution of sterile food. Diarrhœa is to be restrained by suitable astringents, and means taken to improve the nutrition of the child by general hygienic measures, cod-liver oil, maltine, etc. Strict directions should be given to the mother to ensure the proper ventilation of the nursery, the disinfection of stools, the leading of an out-of-door life in a suitable locality, and, in fact, the case should be regarded as one of grave tuberculous disease, and treated on the general principles now finding so much favour in the modern treatment of consumption.

(2) *Ulcers of the Duodenum* occur as:—

(*a*) Acute primary ulcers; and

(*b*) Chronic secondary ulcers.

(*a*) *Acute primary ulcer* of the duodenum has the same pathology as acute gastric ulcer, and the only points necessary to consider in connection with it are the modification of symptoms and sequelæ in virtue of its different site.

(*b*) Chronic secondary ulcer of the duodenum occurs most frequently after severe burns, and these, indeed, constitute the most important group of this class, but they also, though rarely, occur in portal congestion, specific fevers, and in septicæmia. It is not understood why ulcers occur in the duodenum after severe burns, nor is the rationale of their production explained. As in gastric ulcer, peptic digestion is rendered possible by the lowered nutrition of a limited area of the mucous membrane, and several theories have been advanced to account for this. Septic emboli in the duodenal vessels are said to occur, but have never been demonstrated microscopically, and artificial emboli experimentally introduced into the circulation rarely affect the stomach and duodenum, but when affected they are equally so, whereas ulcers following burns occur more frequently in the duodenum than in the stomach. Hunter has shown that when toluylidiamine is subcutaneously injected in the dog, it is eliminated by the bile, and causes duodenitis and ulceration of the duodenal mucous membrane. He therefore suggests that toxic irritating products absorbed from burns are eliminated by the bile and cause similar lesions. Against this theory is the fact quoted by Fenwick that similar lesions occur if the bile-duct is ligatured, and also that after burns ulcers occasionally occur in the stomach. Burns, it is known, are accompanied by inflammation of the mucous membrane of the bowels, and also by gastritis. This may be sufficient explanation of duodenal ulceration, but Fenwick suggests that the stomach and intestines may eliminate poisonous septic products which act as irritants, in the same manner as urea injected subcutaneously appears in the stomach and is found as crystals. The same authority quotes an experiment in which he injected albuminose prepared from septic sloughs of burns, and by these means produced "appreciable duodenitis."

Situation.—The most usual site of duodenal ulcer is between the opening of the bile and

pancreatic duct and the pylorus, on the anterior and mesial side of the duodenum. These ulcers diminish in frequency near the bile duct, probably because peptic digestion ceases after contact with bile or pancreatic juice.

Symptoms.—For convenience of arrangement the symptoms of both classes will be first considered, and their distinctive characters subsequently. Duodenal ulcers generally produce obscure symptoms. Pain varying in character from uneasiness to violent and prolonged suffering occurs in more than half the cases. It occurs two to five hours after food, is situated well to the right of the middle line of the abdomen in the right hypochondrium, and is frequently relieved by sitting up with the body bent forward. The explanation of its occurrence two to five hours after food is that the contents of the stomach reach the duodenum at this period and cause pain by irritation of the ulcer.

Vomiting is much less than in gastric ulceration, and bears no relation to the severity of the pain. Hæmorrhage from the ulcer may be observed either as hæmatemesis or melæna. Fenwick found that hæmatemesis occurred alone or with melæna in an equal number of cases, and that melæna occurred in seven out of seventeen cases where there was evidence that the ulcer had bled. Perforation followed by peritonitis occurs in more than half the cases (53 per cent), and is accompanied by the same symptoms as perforation by a gastric ulcer, and death by general peritonitis is a frequent result. The peritonitis has, however, a better chance of becoming localised in duodenal than in gastric ulcer, for the escaping contents are poured first into the liver pouch described by Rutherford Morison, and may become localised there by adhesions. Perforation of ulcers of the duodenum may also result in a peri-duodenal abscess, or the ulcer may perforate into a neighbouring coil of bowel.

Of the differential symptoms, the following are noteworthy :—

Acute primary ulcer of the duodenum, which is a rare disease, occurs in contradistinction to gastric ulcer much more frequently in adult men than in young women. It usually gives rise to no marked symptoms until sudden melæna, hæmatemesis, or perforation declares its presence. It should be noted in this connection that if hæmorrhage occurs in duodenal ulcer, it is likely to be profuse, because the blood-vessels eroded are usually large trunks (pancreatico-duodenal or gastro-duodenal), and that perforation is very liable to take place on account of the thinness of the duodenal wall. Perforation followed by peritonitis is said to occur in 90 per cent of the cases. Fenwick says it occurred in all his cases.

Secondary ulcers following burns commence a few days after the burns, develop slowly, and reach their maximum in the second week. It is not uncommon to find a small quantity of blood in the dejecta of cases of burning, but this does not necessarily imply ulceration. The enteritis which accompanies many cases is in itself sufficient to explain this. Melæna and hæmatemesis occur in 30 per cent of the fatal cases, and bleeding, which generally occurs from the pancreatico-duodenal artery, is often fatal. Perforation is rare because the ulcer is indurated, and adhesions to the neighbouring tissues have had time to form. The tendency of these ulcers is to cicatrise, and a scar with subsequent stenosis or a pouch of the duodenum may result.

Diagnosis.—Diagnosis is frequently difficult and often impossible. Duodenal ulcers may be distinguished from gastric ulcers by the following points :—

(*a*) Duodenal ulcer occurs much more frequently in men than in women, and rather later in life.

(*b*) Pain is less severe and occurs two to five hours after food; it is referred to the right hypochondriac region, and seldom to the back.

(*c*) Hæmorrhage is very frequent, and melæna with hæmatemesis is most suggestive of duodenal ulcer.

(*d*) Vomiting is independent of food and does not relieve the pain.

(*e*) Perforation is extremely common in acute primary ulcers.

The symptoms of duodenal ulcer may simulate gall-stone colic, but the occurrence of melæna and a hæmatemesis would serve as distinguishing features, and if jaundice was absent in hepatic colic, the gall-bladder should form a palpable tumour.

Treatment.—Except in the chronic forms of ulceration from burns, where hæmorrhage has occurred, drugs are useless, but in this instance some reliance may be placed on half-drachm doses of turpentine given in a gelatine capsule or in milk. The administration of ergot by the mouth, or the hypodermic injection of ergotine, might also be tried. If the symptoms are not urgent and point to cicatrisation of the ulcer, strict rest in bed, the blandest of food, or the substitution of rectal feeding, should be enjoined. If there is reason to suppose that perforation has taken place, or if hæmorrhage has occurred more than once and is exhausting the patient's strength, prompt abdominal section offers the best prospect, as by this means there is a fair chance that the perforation may be discovered and general peritonitis prevented, or the bleeding vessels may be secured.

(3) *Syphilitic Ulceration.*—Syphilitic ulceration rarely occurs in the intestine. Ulceration and stricture, condylomata and mucous patches are well known in the rectum, and various pharyngeal lesions constitute well-marked features of syphilis, but the proximal and distal ends of the digestive tract are almost exclusively affected. In infantile syphilis ulcerative plaques and small

gummata have been found, but their presence as a clinical feature is not established, a remark equally applicable to acquired syphilis. In severe and neglected cases of syphilis, with gummata elsewhere in the body, diarrhœa occurs, and may be due to broken-down gummatous ulcers in the intestine. In such circumstances before arriving at a diagnosis of ulcer the question of lardaceous disease of the intestine, and the possibility of tuberculous ulceration in a syphilitic subject, must be excluded. The treatment of syphilitic ulceration of the intestine is that of the general disease.

(4) *Catarrhal or Follicular Ulcers.*—These are found in inflamed conditions of sufficient severity of the intestinal mucous membrane, and are more common in the large than in the small intestine. They occur either as simple erosions of the mucous membrane apart from the intestinal glands, or as the result of inflammation and destruction of the solitary, Lieberkühn's, or Brunner's glands. Both varieties are usually present, it may be in large numbers, in the more severe cases of enteritis. Their presence is usually indicated by blood in the stools. Small ulcers are also found above the site of intestinal obstruction, but the pathology of them is still doubtful. The treatment of follicular and catarrhal ulcers is that of enteritis.

(5) *Malignant Ulcers* (*vide under* "Malignant Diseases of the Intestine," p. 533).

(6) *Hæmorrhagic Ulcers.*—In Bright's disease hæmorrhage into various organs is liable to occur, and in the intestine the chosen site is under the mucous membrane. This submucous hæmorrhage instead of being absorbed may break down and form an intestinal ulcer.

Ulcers of the intestines in Bright's disease are usually not single but multiple. As enteritis is not an uncommon complication of kidney disease, it is therefore questionable whether the intestinal ulcers of Bright's disease should be regarded as broken-down submucous hæmorrhages or as catarrhal ulcers occurring in an enteritis complicating the original disease. Both varieties probably occur, and it is also probable that in purpura, scurvy, anæmia, etc., intestinal ulcers of purely hæmorrhagic origin develop.

(7) *Vascular ulcers* are either venous or arterial. Whether venous or arterial they depend on defective blood-supply to an area more or less limited, this leading to necrosis of the intestinal mucous membrane and the subsequent formation of an ulcer.

Venous ulcers occur in cirrhosis of the liver where the portal circulation is impeded, and probably have the same pathology as ulcers of the leg connected with defective venous circulation.

Ulcers due to arterial blocking are described. The artery may be occluded in either of the three following ways :—

(*a*) Plugging of the mesenteric vessel or one of its branches by emboli. This variety may complicate a case of endocarditis. The symptoms would probably be intense abdominal pain and tenderness coming on suddenly, followed, if the patient lives long enough, by bloody stools, tympanites, and peritonitis.

(*b*) The mesenteric artery, or one of its branches, may be the site of thrombosis ; or

(*c*) Atheroma may obstruct the lumen, and circulation becomes so defective that numerous ulcers of the mucous membrane result. The pressure of a tumour on a vessel may effect the same result.

Lardaceous Disease.—The intestine is affected by lardaceous disease next in frequency to the kidneys, spleen, and liver. If it does occur at all, it is extremely rare for the intestine to be the only structure involved ; indeed, they do not usually become waxy until the disease is far advanced in the solid organs. The intestine in 30 to 40 per cent of cases of lardaceous disease is affected.

Morbid Anatomy.—As in other organs the amyloid changes commence in the small arteries. In mild cases the change is confined to the mucous and submucous coats, the villi being specially affected, but all coats may become involved. To the naked eye the mucous membrane presents a glossy translucency, is pale and somewhat swollen, but the iodine test is usually required to settle the question, when the characteristic reaction will be found as spots and lines distributed irregularly over the surface. If the waxy disease is advanced, the mucous membrane may break down and form small ulcers. The small intestine, especially the lower end of the ileum, is the most usual seat of the disease.

Symptoms.—The most prominent symptom is diarrhœa with or without hæmorrhage. The occurrence of a persistent diarrhœa is usually the first indication that the intestine has become waxy, and the occurrence of this symptom in a case where other organs are known to be waxy is strong evidence of this. If the stomach is also waxy vomiting may occur, but the possibility of the vomiting being uræmic should always be remembered. Abdominal pain and tenderness are rare.

Diagnosis.—It is always difficult and often impossible to distinguish lardaceous disease of the intestine from tubercular in a given case where either or both may occur, as in the later stages of phthisis. It is only by consideration of each case in all its bearings that an opinion can be formed.

Treatment.—If possible the causative disease should be removed, *e.g.* syphilis, tubercle suppurating joints, etc. In this the only hope of cure lies. In the great majority of cases all we can hope to do is to restrain the diarrhœa by astringents, etc. (*vide also* "Lardaceous Degeneration," vol. v.).

Malignant Disease of the Intestine.—Malignant growths of the intestines differ but slightly from malignant disease of other parts, in so far as the characteristics of the growths are concerned, but when a hollow tube is attacked the potency of every part of which is recognised as important, and when the dependence of health on a vigorous digestive apparatus is remembered, the special importance of this subject will be admitted.

Site.—Of 1148 cases collected by Ewald—

- 874 occurred in the rectum.
- 148 occurred in the large intestine (of these twelve occurred in the transverse colon).
- 64 in the cæcum, including the appendix.
- 26 in the ileum.
- 19 in the duodenum.
- 17 in the jejunum.

In the above Table, cancer of the small intestine (duodenum, jejunum and ileum), as compared with the large intestine (cæcum, appendix, colon, sigmoid flexure and rectum), occurs in the proportion of 1 to 17½. It will thus be seen that the large intestine is much more frequently affected than the small, and that the jejunum is the portion least likely to be involved. Statistics of other authors show the jejunum to be more rarely involved than the above. It is a noteworthy fact that where the intestinal contents stagnate longest (rectum), or pass through a part with diminished calibre, or where two differing surfaces adjoin (ileo-cæcal valve and pylorus), there cancer is most frequently found.

Pathological Anatomy.—Carcinoma of the intestine may be primary or secondary. Primary is far more common than secondary cancer, which, indeed, is rare, and occurs either by metastasis, or by extension from neighbouring organs (pancreas, uterus, stomach, etc.), and being but a complication of pre-existing disease, requires no special mention here. Primary cancer of the intestine occurs as cylindrical-celled epithelioma, more rarely as a spheroidal-celled growth. Either of these forms may undergo colloid degeneration and produce a third variety known as colloid cancer.

Cancer of the intestine begins as a small deposit in the mucous membrane, usually in Lieberkühn's glands. As it increases in size it spreads in such a manner as to present itself in three distinct aspects, namely:—

1. As a nodule, which, expanding equally in all directions, becomes a more or less rounded tumour, the intestinal surface of which is apt, from irritation of the intestinal contents and bacterial invasion, to become ulcerated and assume a polypoid outline.

2. The growth may spread laterally rather than towards the lumen of the bowel, and assume the form of a plaque or raised flattened mass on one side only of the intestinal wall, with well-marked edges, and often ulcerated centre.

3. Most commonly extension takes place in an annular manner, so that the intestine is surrounded by a ring of cancerous deposit often of small longitudinal depth. This variety is the commonest cause of malignant stricture. It occurs most frequently in the large intestine, and is a frequent cause of intestinal obstruction. In the later stages ulceration at the site of growth, dilatation of the bowel above the stricture, ulceration at or above the stricture, and probably hæmorrhage, mark the subsequent course. Extension early takes place to lymphatic glands, and secondary deposits occur usually in the liver, and other neighbouring abdominal organs, the peritoneum becoming invaded by direct extension; or ulceration and perforation may occur into the bladder, stomach, and other parts of the intestine, or into the peritoneum, generally or becomes localised by adhesions. In some cases of annular stricture gangrene of the cancerous deposit is apt to occur. The author has seen a case of annular stricture of the sigmoid which caused intestinal obstruction, necessitating colotomy. The case ended fatally by peritonitis, due to gangrene of the annular stricture with subsequent perforation. The stricture was so insignificant in size that it was impossible to say definitely without microscopic investigation, and the corroborative and extensive secondary deposit in the liver, that it was a malignant one.

A few special points are noteworthy in cancer of the duodenum, which is usually columnar-celled, but may be spheroidal-celled. The growth begins in the mucous membrane, or possibly in Brunner's glands, though the latter origin is doubtful. A malignant growth of the head of the pancreas and of the lower end of the bile and pancreatic ducts or the biliary ampulla is an extra-duodenal growth, in which the duodenal mucous membrane is not, until the last stage, and may not be at all, involved. Any part of the duodenum may be the site of primary cancerous growth, but it is important to distinguish from a clinical point of view whether the original site is above or below the biliary papilla. Malignant growths of the duodenum, as elsewhere in the intestine, have a tendency to produce annular stricture resulting in dilatation of the duodenum and stomach behind the stricture, and the symptoms simulate those of pyloric stricture.

Symptoms.—The presence of a malignant growth in the intestine may be revealed in a variety of ways, but a diagnosis in the early stages is rarely possible. The initial subjective and objective symptoms seldom occur until the growth has existed for some time. It may happen that acute intestinal obstruction will be the first sign to arouse suspicion, a condition of affairs explicable, and caused by the sudden

blocking of the intestinal lumen at the site of growth by hard scybala, or foreign bodies. Perforation of the bowel with peritonitis may be the first intimation of serious mischief. The symptoms, however, generally develop slowly. Vague dyspeptic symptoms, a sense of discomfort in the abdomen, even amounting to pain, constipation alternating with diarrhœa, a distaste for food, mental irritability, nausea, vomiting and progressive loss of strength, with cancerous cachexia, usually precede the more definite symptoms of intestinal blocking. The assertion that if a patient over thirty-five years of age complains of persistent dyspeptic symptoms for the first time, cancer of the stomach should be suspected, is equally applicable to cancer of the intestine, with perhaps this addition: that the sufferer from intestinal cancer will probably also complain of flatulence and diarrhœa alternating with constipation. Unfortunately, the early symptoms of cancer, until it has attained to the stage of a palpable tumour, are always vague and uncertain. If a tumour of the small intestine can be detected, it may occupy any part of the abdominal cavity, because, with the exception of the second and third part of the duodenum, and the colon, the intestine is attached to a longer or shorter mesentery, which allows of considerable displacement by the weight of the tumour itself, or by pressure communicated to it from without. The tumour may vary in size, or be more readily felt at one time than another, due to the amount of abdominal distension or presence of fæcal masses behind a stricture. The stools frequently contain blood, and, indeed, considerable hæmorrhage may take place from the ulcerated mucous membrane. For further symptoms of intestinal obstruction in Malignant Disease, *vide* p. 540.

In cancer of duodenum, the general symptoms irrespective of the part affected are wasting, the gradual appearance of cancerous cachexia, anorexia, nausea, and vomiting at variable intervals after meals, pain in the right hypochondrium, hæmatemesis, rarely melæna, and in the later stages signs of dilatation of the stomach from stenosis of the duodenum, will usually be present. In the later stages, after ulceration of the mucous membrane has allowed of auto-intoxication from bacterial absorption, the temperature rises. If the growth invades the duodenum above the biliary papilla, which usually enters the duodenum three or four inches below the pylorus, the vomited matter is unlikely to contain bile, and the symptoms are similar to those found in cancer of the pylorus. If the growth is below the biliary papilla, bile and pancreatic juice regurgitate into the stomach, and their presence in the vomit can be easily recognised. Bile may also in this case be deficient in the stools. In supra-papillary cancer the tumour is movable, and resembles a pyloric cancer, whereas in the infra-papillary variety it is fixed, and may be mistaken for cancer of the head of the pancreas. The growth may occur at or so near to the biliary papilla itself as to produce symptoms indistinguishable from those brought about by cancer of the common duct, or its ampulla, where jaundice is an early symptom.

Diagnosis.—Diagnosis is never easy, and in the early stages, in the absence of a palpable tumour without an exploratory laparotomy, it is impossible. If an abdominal tumour is absent, the persistence of symptoms in spite of treatment, the age of the patient, progressive loss of weight, and the appearance of cancerous cachexia, are the chief symptoms to rely on, but it is often necessary in the early stages to arrive at an opinion by a process of exclusion. After the appearance of a tumour great care has still to be exercised before a definite opinion can be formed.

Cancer of the first part of the duodenum is practically indistinguishable from cancer of the pylorus, but differs from simple ulcer of the stomach by the presence of a tumour, by the small quantity of blood vomited, and by pain occurring some hours after food.

Malignant disease of the duodenum below the pyloric papilla is distinguished from cancer of the head of the pancreas or of the lower end of the bile-duct by the absence of jaundice and by the presence of signs of duodenal stenosis and gastric dilatation.

If the case is one affecting the intestine below the duodenum, we must first ascertain by the administration of enemata and purgatives whether, after all fæcal accumulation is removed, any tumour is left. In every case rectal examination is then made, and in women, if possible, vaginal also. The history of the case and careful weighing of the symptoms may aid in the differential diagnosis from other conditions producing abdominal tumours, especially malignant disease of the uterine appendages, hydronephrosis, floating kidney, and tuberculosis of the peritoneum, and from other causes of intestinal obstruction, such as healed typhoid and tuberculous ulcers, and the impaction of gall-stones.

Treatment.—Medical treatment can only be palliative. Surgical operation should receive the first consideration, because a radical cure of malignant intestinal growths is now no longer impossible. The diet should be so regulated that no irritating matter is allowed and little fæcal residue results. It should be chiefly fluid, easy of digestion and absorption. By this means the chance of intestinal obstruction is diminished, pain is lessened, a greater degree of rest is secured, and the tendency to hæmorrhage from the ulcerated mucous membrane is obviated. In advanced cases it may be advisable to rely largely on rectal feeding. In the last

stages of the disease, when all hope of recovery has passed and the patient's life is rendered miserable by constant suffering, opium or morphia should be regularly given in sufficient doses to secure relief and peace.

In cancer of the duodenum with symptoms of stenosis and dilatation of the stomach, lavage gives temporary relief, and gastro-enterostomy will permanently relieve the distressing vomiting and prolong life. Intestinal antiseptics, such as salol, appear to do good, if fever and other symptoms of auto-intoxication are present. For hæmorrhage, turpentine or ergot may be tried.

In cancer of the small or large intestine, excision of a limited growth offers the chance of a radical cure, and is in any case the best palliative. Excision of growths of the large intestine has given the best results, and exploratory operations are advisable as soon as there is a reasonable doubt of their presence. If the disease has advanced too far to admit of excision of the growth, an intestinal anastomosis performed at the situation most suitable to the particular case will allow of a temporary return to health and remove the obstructive symptoms (*vide* "Intestinal Obstruction," p. 552).

Sarcoma of the Intestine.—Sarcoma of the intestine is more rare than carcinoma. Few cases have been reported, and it is difficult to estimate exactly how many of the reported cases are true sarcomata, sarcoma, lympho-sarcoma, and lymphoma being confused in the descriptions. Lympho-sarcoma occurs in the intestine in connection with Hodgkin's disease, and may grow to a large size. Sarcoma here, as elsewhere, may occur at an early age, and cases have been reported in children of a few years of age. The infiltration of the bowel rarely produces stenosis or obstruction; on the contrary, the lumen is usually made larger by the growth. Constitutional symptoms and wasting appear early, and death occurs after a shorter illness than in carcinoma. The prognosis and the results of treatment are equally bad.

Non-Malignant Tumours of the Intestine.—*Adenomata.*—Adenomata arise from Lieberkühn's or Brunner's glands, and have a typical acinous structure. A considerable amount of fibrous tissue may be found in their matrix when they are fibrous, or they may be myxomatous and soft. They may be attached to the mucous membrane by a broad base or by a narrow pedicle of variable length. The large intestine is their most frequent site, and considerable numbers may be present.

Fibromata.—Fibromata arise from the connective tissue of the submucous coat, are always numerous, usually small, and may be either sessile or pedunculated.

Myomata and Fibromyomata arise from the muscular coat, are usually small, and possess a pedicle.

Lipomata and Angeiomata also occur, but are rare.

All the above are usually classified as intestinal polypi. Clinically these innocent tumours may give rise to no symptoms, or to irregular attacks of diarrhœa, accompanied by hæmorrhage.

Their weight may cause invagination or intussusception, and if large, they may produce mechanical obstruction. Cases have been recorded where they have become detached and passed per rectum, making the diagnosis clear.

Actinomycosis of the Intestines is a rare disease. One mode of infection, *i.e.* of the deposition and growth of the essential fungus, is through the intestinal mucous membrane. The lesions may occur in any part of the intestine, but recorded cases point to the large intestine as the most usual site, the cæcum and appendix being probably the most frequent. The early lesions consist of small abscesses in the submucous coat, apparent to the naked eye only as indefinite semi-translucent swellings, which, when punctured, contain pus and the small spherules of the fungus. These abscess cavities either rupture into the intestine and form an ulcer, which may heal and leave a puckered cicatrix, or, more usually, infiltrate and destroy the muscular coat, acting as foci of a spreading chronic inflammatory induration, which extends to the peritoneum and causes matting of the surrounding parts, interspersed with small abscess cavities. A palpable tumour is thus formed, which may, and usually does, run a chronic course, tends to involve the abdominal wall and to open externally, but when it does so, fails to discharge pus in quantity like an ordinary abscess. In the discharge the characteristic granules are found. The process is essentially a chronic spreading inflammation with suppuration, which may, by virtue of its situation by extension or metastasis, spread to the liver, kidneys, etc., or may by a fistulous opening communicate with the bladder, stomach, or other part of the intestine.

Symptoms are always obscure, and many cases remain doubtful or unsuspected until a necropsy reveals the true nature of the disease. In the early stages the symptoms are usually those of intestinal irritation and atony—diarrhœa of variable severity and abdominal distension from flatulence. As the disease advances a palpable tumour becomes evident. According to the situation this will resemble an appendix abscess, a sarcoma, etc., and unless it is possible to find granules in the stools, urine, or discharge of the abscess, it is exceedingly difficult to form an opinion as to the nature of the case. Temperature is rarely elevated, and the course of the disease is strikingly slow unless some complication such as a perforation and peritonitis should supervene.

Diagnosis.—Careful search must always be

made for the characteristic spherules of the fungus in the stools, in the discharge of abscess if the case has arrived at this stage, and in the urine. On the detection of these only can a positive opinion be formed. Ransom says—"When a subacute or chronic inflammation occurs which spreads without much elective affinity for any tissue or viscus, which is hard, even woody, to the touch, involves the skin, softens in the centre slowly, at length yields pus in scanty proportions relative to the bulk of the swelling, and does not heal when freely opened and drained, it is almost certainly an infective malady, and actinomycosis may be strongly suspected, and the spherules and colonies should be sought for."

Treatment.—Large doses of iodide of potassium, at least one drachm daily, is the most hopeful remedy, and, it is said, may cure. Ransom reports such a case. Intestinal antiseptics also afford relief. If a palpable tumour exist it should be freely opened and drained. If circumstances permit, it is possible that scraping or any means to eradicate the then apparent lesion are justifiable, in spite of the fact that other organs or tissues may be involved. (*Vide* also "Actinomycosis" and "Appendicitis.")

Intestines, Surgical Affections of.

Intestinal Obstruction

Intestinal obstruction is caused only by mechanical obstacles, and surgical consideration is demanded from the outset. A sudden complete obstruction of any part of the small or large intestine causes the same serious symptoms as are met with in strangulated hernia, and the pathological conditions produced above the obstruction (and these are far the most important) are the same in both. It is therefore obvious that if as early surgical treatment could be adopted in cases of intestinal obstruction as in cases of strangulated hernia, the present bad results, which every surgeon deplores, would at once improve. The difficulty of early diagnosis, more than anything else, contributes to fatal delay. A careful and intelligent practitioner can scarcely make any mistake when he sees and feels a strangulated hernia, but the diagnosis of a similar condition hidden in the abdomen is surrounded by difficulties.

For the first few hours in a case of sudden complete obstruction, the symptoms produced are those brought about by any condition which may cause a profound impression on the abdominal nerves, and are those of abdominal shock. Many serious abdominal lesions, such as perforated gastric or duodenal ulcer, ruptured vermiform appendix, leaking ectopic gestation, gall-stones, etc., are attended by symptoms which are indistinguishable from those occurring in the early stage of sudden obstruction. That they are due to nervous influence in cases of obstruction is proved by experiments on animals, which show that vomiting and other acute manifestations do not follow artificial constriction of the intestine when the mesenteric nerves supplying it have been previously divided. This knowledge also helps to an understanding of the marvellous relief following the use of opium.

The second stage soon follows on the first. Shock is recovered from, and the typical symptoms and signs of obstruction are now more likely to be met with than at any other time, if carefully looked for. These are produced by a vigorous attempt on the part of the intestine to get rid of the obstruction by forcible contractions in its unstriated muscular coat. The forcible contraction of unstriated muscle, here as elsewhere, induces the most agonising pain, in great part intermittent, and many patients already more or less familiar with intestinal pain, as most of us are, will at this stage express a confident belief that if only wind could be voided they would soon be well again. Shortly, and not slowly, the vigorous contractions cease; the intestines become more distended, gas and fluid increasing, and the third stage of intestinal paralysis is reached. This is accompanied by venous engorgement in the intestine above the obstruction, most marked in its immediate neighbourhood, but rapidly involving higher and higher portions, due to interference with the blood circulation by distension. Following the circulatory disturbance there is an increased quantity of fluid poured into the intestine, absorption being at the same time checked. Increased gas formation from the putrid contents helps to further the distension. Free fluid is also found in the peritoneal cavity, and in the most acute cases may contain suffi-

cient blood to colour it, as occurs in the sac of an acute strangulated hernia. The intestinal wall above the obstruction becomes red, thickened, sodden, and friable, and the first histological alteration apparent in it is an active destruction of the cells lining its mucous membrane, which play so large a part in its protection from septic invasion. Below the obstruction the bowel is pale and contracted. The bacterium coli now becomes exceptionally active, thriving in the stagnating intestinal fluids and invading the intestinal walls. The transition from this stage to the final one, if the patient survives the auto-intoxication produced by absorption from his own intestines, and exhaustion resulting from the starvation, pain, and vomiting, is by peritonitis, brought about either by the migration of organisms through the distended and damaged intestinal walls, or by the more gross method of perforation through a distension ulcer or gangrenous patch above the obstruction. In the final stage the only diagnosis possible may be that of peritonitis.

Classification

No classification is entirely satisfactory. Perhaps the best is one which divides the causes of mechanical obstruction into five classes:—

1. Strangulation of the intestine by bands, accidental rings, internal herniæ, adhesions.
2. Defects of position in parts of the intestine, such as intussusception, volvulus, kinks, displacement by tumours, etc.
3. Blocking of the lumen by foreign bodies, polypi, hard fæcal masses, etc.
4. Strictures, simple and malignant.
5. Paralytic ileus.

1. *Strangulation by Bands, Internal Herniæ, Adhesions, etc.*—Strangulation by bands appears to have become more common since abdominal section ceased to be a rare operation, for many of these bands result from a preceding peritonitis, and may be caused by the traumatism of operation. In addition to this cause, disease in the appendix or in the pelvic viscera, or tuberculous lesions in the abdomen, are frequent sources of a peritonitis which leaves adhesions to be stretched and thinned into cords or broader bands. Another origin is not uncommon: a tag of omentum adhering in the neighbourhood of a femoral or inguinal hernia in course of time, by a process of stretching and rolling, is converted into a cord or band, often of a great length. Such cords or bands produced by any of the causes mentioned may strangle the intestine in every conceivable way, so much so that no two cases are exactly alike. Adhering at both ends, either to viscera or parietes, and remaining free in the centre, a bridge is formed under which a loop of intestine may slip and be strangled. A long cord may loop itself and become knotted round an intestinal loop, as a piece of string purposely tied round it would. On the other hand, the intestine may wind itself round such a cord, and become strangled by its own act. More than one case has been reported where two bands have been found post mortem, the wrong one having been divided during operation. In addition to the band, it is not infrequent for a volvulus to be found in the ensnared intestine beyond the band. Many of the well-marked cords are not, however, due to a preceding peritonitis, but have a congenital origin from persistent remnants of the omphalo-mesenteric vessels. In this connection, too, the Meckel's diverticulum may be noticed. In the human fœtus, about the eighth week of intra-uterine life, the vitelline canal normally disappears. If it does not, the connection between the small intestine, usually at the convex surface of the ileum, and not far from the ileo-cæcal valve, and the umbilicus, may permanently remain. Then a tubular off-shoot or diverticulum possessed of the same structures as go to form the small intestine may be found extending from the intestine to the umbilicus. More commonly a portion of this tube is obliterated, and there is found projecting from the intestine a short diverticulum attached by a cord to the neighbourhood of the umbilicus. At times the distal extremity of the diverticulum, or its obliterated representative, is set free and may continue to float, or may contract fresh adhesions to any part of the parietes or viscera. Another band producing precisely similar results may be constituted by the adhesion of normal structures, for example the vermiform appendix, the Fallopian tube, the appendices epiploicæ, the intestine itself, and its mesentery. Cases also are recorded of intestine strangulated by the long pedicle of an ovarian cyst.

The pathological results produced in the intestine by strangulating bands are of two kinds:—(1) Those limited to the area of intestine strangulated; and (2) changes in the intestine above. The latter are similar in every instance of acute obstruction, and have already been referred to. The local changes are those produced directly by the constricting agent, causing necrosis from pressure at the site of constriction, and vascular changes in the gut beyond, which tend to produce gangrene of the portion ensnared. An early release of the gut shows a pallid depression at the point constricted, the pallor and constriction disappearing in a few minutes after its release. At a later stage the pallid constriction does not disappear, and section of the intestine shows that the whole thickness of its wall, except the peritoneal coat, has been destroyed, the site of the constriction internally being marked by ulceration of the mucous and muscular coats. This ulceration is found in the entering portion of gut, and at the limit of the distended portion when the constriction is a small tight band, and only a few inches of gut have passed under it, as is generally the case

(Fig. 1); but not uncommonly it occurs at the further limit of the ensnared gut, as when a large coil of intestine passes under a loose band, and is obstructed only at its outlet (Fig. 2). Evidences of local peritonitis are soon seen in the strangulated loop. Its surface becomes dull and sticky, and later on is covered with purulent lymph. Finally, the signs of gangrene appear. The lowest portion of the ileum is most commonly caught in this form of obstruction.

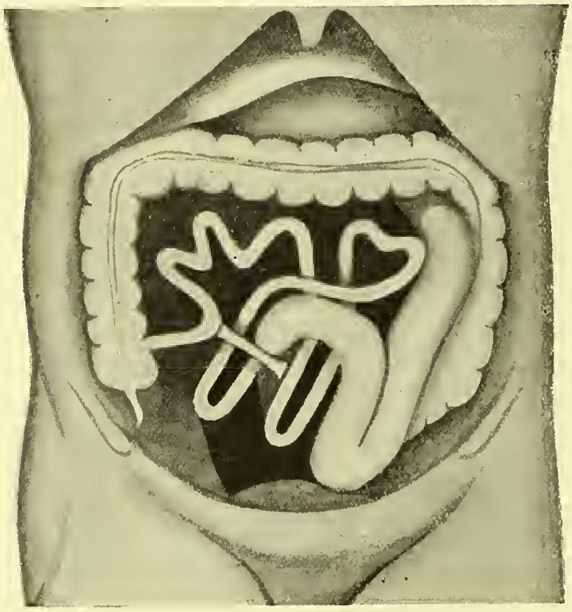

Fig. 1.—Strangulation by tight band.

Internal Herniæ.—In the peritoneum, covering the posterior abdominal wall, chiefly at the duodeno-jejunal and ileo-cæcal junctions, and in relation to the cæcum and sigmoid flexure, pouches of peritoneum are normally found. Into one of these a portion of bowel may enter. Indeed, cases are recorded in which the whole of the small intestine has been found in such a sac. The gut may be held and become strangulated there; or a loop of intestine may pass through either normal or abnormal openings in the mesentery or omentum, such as are occasionally found in healthy subjects, or such as may be left after tears from injury, or the foramen of Winslow may allow the gut to pass through and become retained in the lesser peritoneal sac, strangling it at its entrance; or as the result of a congenital or accidental opening, intestines may pass into the pleura through the diaphragm and be nipped by the congenital or acquired orifice. Diaphragmatic hernia is practically limited to the left side, as congenital openings are generally found there, and the liver protects the right side from the effects of injury.

Adhesions.—Adhesions occur often as a result of preceding peritonitis, due chiefly to tuberculosis, appendicitis, and, in women, to pelvic lesions. In the latter case the results are somewhat different from those produced by the ordinary bands or adhesions. A loop of intestine becomes adherent to some portion of the pelvic wall, frequently the bladder or uterus. Under the bridge of intestine and mesentery so formed, coils of intestine find their way between the arch of intestine and mesentery in front and the pelvic wall behind. The intestinal contents pass freely into the ensnared loops, but have difficulty in making their escape. Distension follows, and the increased tension brought about by this forces the exit channel into a position from which it cannot escape, usually the sharp angle formed by the mesentery, and this portion of intestine becomes acutely strangulated as by a band (see Fig. 2). Kinking of the intestine may cause obstruction by the dragging of a limited adhesion (Fig. 3), producing a valvular occlusion, or so much matting may attend extensive adhesions that the passage of intestinal contents is prevented.

2. *Defects in Position of Parts of the Intestines.*—Intussusception is the most common example of this variety, and one of the frequent causes (about one-third of all cases) of intestinal obstruction. A portion of constricted bowel is received into the cylinder below it, so that a transverse section through the involved portion

Fig. 2.—Strangulation by loose band.

from without inwards would show (1) the sheath with its mucous surface in contact with the mucous surface of (2) the returning or middle layer, the peritoneal coat of which is in contact with the peritoneal coat of (3) the entering

layer. The inner two layers are known as the intussusceptum, the outer or sheath as the intussuscipiens. The upper part is known as the neck or collar, and the apex is the lower extremity of the intussusceptum. The mesentery is found between the layers of the intussusceptum, and assists in the arrest of further progress of the involved portion of intestine by the traction it exerts. In addition to this the intussusceptum is made to take a curved form by it, and the orifice at its apex to become more slit-like than circular. Any part of the small or large intestine may be involved, and these varieties are described under the name of the portion of intestine affected.

Intussusception in children before they are five years of age is common. It appears to follow frequently the vigorous, irregular peristalsis caused by purgative medicine or irritating foods, and is seldom due to any organic disease. In adults it is comparatively rare, and with few exceptions is occasioned by growths in the bowel, either polypi or annular malignant growths. The ileo-cæcal valve certainly plays a very important part in its production, as the ileo-cæcal junction is the commonest site of it. This valve has been aptly compared to the anus, and intussusception here to prolapse of the rectum. I have seen acute intussusception follow immediately on severe strain in a healthy young adult, and in babies, too, vigorous jumping up and down previous to the attack has been observed in so many cases that traumatism must be noted as an exciting cause.

Apart from the intussusceptions found post-mortem, which produce no obstruction and cause no symptoms, and appear in the act of dying, two distinct varieties of intussusception, pathological and clinical, are met with: the acute and the chronic. In acute cases the intussuscepted bowel and its mesentery are nipped by the collar at the neck, and the intussusceptum is affected as is the imprisoned bowel in a strangulated hernia: First, The venous circulation is obstructed in the intussusceptum; then follows œdema and consequent interference with the arterial supply; next the damaged tissues yield to the attack of the bacillus coli, and become inflamed, gangrene occurring as the final result. Above the intussusception the intestine is distended, and destructive circulatory changes are brought about in it as in all obstruction cases. The most important pathological facts to bear in mind from the clinical point of view are the swelling of the intussusceptum produced by venous engorgement, which in the course of a few hours may be so pronounced as to make reduction impossible. The engorgement is most marked at the apex of the intussusceptum, which becomes so swollen, rounded, and firm as to resemble a tumour, and to effectually resist ordinary efforts made for its release. Of even greater importance is the fact that adhesions are likely to form rapidly in acute cases, and to glue together the peritoneal coats of the involved intestine. These adhesions, though favourable to spontaneous recovery by sloughing of the intussusceptum, are a hindrance to its surgical reduction. The usual causes of death are gangrene of the sheath, perforation in it or in the bowel above, and gangrene of the intussusceptum.

In the chronic variety there is at first little if any interference with the vascular supply of the involved bowel, and inflammation is consequently not brought about. Even after many months in some chronic cases reduction has been easily effected, on account of there being but a small amount of swelling and an absence of inflammatory adhesions to glue the displaced and opposed peritoneal surfaces together. Sooner or later, however, in these cases more active mischief develops. Frequently, in the neighbourhood of the lesion, ulcers form, and perforation with peritonitis terminates existence. The gut above is frequently hypertrophied as in other chronic obstruction cases.

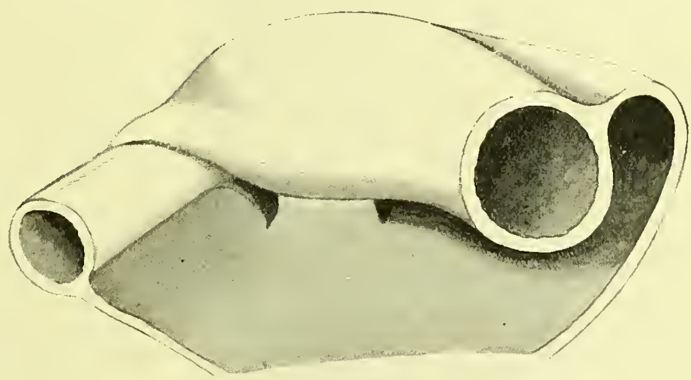

Fig. 3.—Kink with adhesion to a tuberculous mesenteric gland.

Torsions and Volvulus.—Torsions and volvulus, though frequently spoken of as common, are quite rare. With a sufficiently long mesentery the most complicated knots and twists may arise, but except for the puzzling difficulties in dealing with these complicated conditions surgically, they are of small clinical importance.

Volvulus is a well-marked variety. It consists in the twisting of an intestinal loop round an axis formed by its mesentery, a congenital or acquired lengthening of which is commonly found associated with it (Fig. 4). It chiefly occurs in elderly adults, and the sigmoid flexure is its most common site. For this there are several reasons: the sigmoid is a long loop

with its commencement and termination close together. The loop has consequently a narrow neck, and in patients suffering from habitual constipation is filled with heavy matter, which tends to elongate its mesentery.

Volvulus is one of the most acute varieties of intestinal obstruction. By torsion of the mesentery the circulation of blood in the involved intestinal loop is suddenly interrupted. The intestine becomes of a plum colour, and rapidly distends with gas and fluids. Obstruction is caused by the pressure produced by one end of the loop pressing upon the other end.

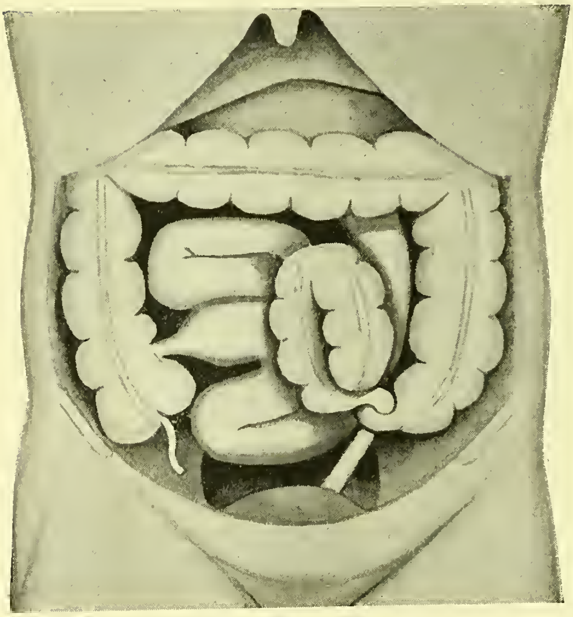

Fig. 4.—Volvulus of sigmoid flexure.

The distension of the sigmoid loop may become so great that the whole abdomen appears to be occupied by it, and the diaphragm may be pushed up, pressing upon and displacing the heart. Organisms soon find their way through the damaged and softened gut, and peritonitis as the result of this transmigration frequently causes death. If the patient lives long enough small patches of gangrene appear in the gut, and by perforation of these rapidly fatal peritonitis may be caused; but this, owing to the speedy fatal issue, is a rare termination. I have recently met with a rare case in which volvulus of the sigmoid was the cause of chronic obstruction.

Kinks.—Kinks are often the result of fixation of an intestinal loop by adhesion, and occur at the junction of the fixed with the movable portion (*see* Fig. 3). At other times they result from the union of two loops, as when a strangulated hernia is reduced and the damaged bowel is retained by inflammatory adhesions in the position held by it in the sac.

The possibility of displacement and compression of the intestine by tumours external to it, though rare, ought to be borne in mind. Tumours of the ovary, of the uterus, of the broad ligament, of the pancreas, of the liver, of the spleen, of the kidney, and inflammatory masses, have all been noted as causes of intestinal obstruction.

3. *Blocking of the Lumen from within.*—The most common cause of this accident is a large *gall-stone,* which usually escapes into the small intestine by a direct fistulous communication between the gall-bladder and the duodenum. It would appear unlikely that a gall-stone small enough to get through the common bile duct could obstruct the intestine. The stone may be arrested at any part of its course through the small intestine, but its usual place of arrest is in the lower ileum. If it passes into the large intestine the dangers are as a rule over, though I have operated on a case of intestinal obstruction produced by a gall-stone blocking and causing inflammation of the sigmoid flexure, and have had to remove from the rectum a large gall-stone which could not pass the anal sphincter, and was causing serious obstruction symptoms. Hard fæcal masses, clumps of cherry-stones, intestinal calculi, and a variety of foreign bodies, introduced either by the mouth or rectum, are also reported as causes of obstruction.

Polypi may be benign or malignant, and single or multiple. Next to the rectum they are most frequently seen in the colon or ileum. Obstruction may be caused directly by the tumour or tumours blocking the intestine, or by their producing intussusception through dragging on their attachments.

4. *Strictures.*—Strictures are simple and malignant, the latter chiefly predominating.

Malignant strictures are most commonly found at and after middle life, and in the large intestine. In the small intestine they are comparatively rare. Not uncommon sites are the ileo-cæcal valve, the hepatic and splenic flexures of the colon, and the sigmoid flexure. Apart from the rectum, they occur with the greatest frequency in the sigmoid flexure (in 60 per cent of cases). The stricture is brought about by an annular growth, sometimes remarkably limited, of cylindrical-celled carcinoma, which contracts the bowel more and more as it grows. Cancerous growths of the bowel wall and growing into its lumen occasionally obstruct it, though not causing genuine stricture, and the same is true of other growths, such as lipomata, fibromyomata, polypi, adenomata, sarcomata, and cysts, all of which, though rare, have been found.

Simple strictures are commonly found in the small intestine, and are comparatively rare in the large. They occur mostly in young people, and their commonest cause is a healed tuberculous ulcer in the neighbourhood of the ileo-cæcal valve. Other causes are: congenital

defects, usually in the portion of ileum occupied by Meckel's diverticulum, when they are probably due to an excess of the developmental involution by which the process is normally obliterated, traumatism, contractions subsequent to ulceration of a portion of strangulated gut, and contractions following syphilitic ulceration, which selects by preference the large intestine. Whether the stricture be simple or malignant, the form of obstruction caused by it is usually chronic, and then dilatation with hypertrophy of the walls of the intestine above the stricture is produced by it, and is an important clinical feature. The cæcum, especially in these cases, may reach such a size as to extend far into the left side. The mucous membrane of the strictured colon is frequently in a catarrhal state owing to the injury produced by putrefaction and mechanical irritation by the fæcal contents of the bowel. This offers an explanation of the frequency with which patients suffering from stricture complain of diarrhœa, which may be the most noticeable symptom. Pouching of the intestine above the stricture is not uncommon, and foreign bodies are frequently found in these pouches.

5. *Paralytic Ileus.*—After abdominal section or the apparently successful reduction of a strangulated hernia, or a severe painful accident, such as fracture of the femur, due to indirect violence, the patient may die in from two to ten days with distended painful abdomen, vomiting, in the end fæcal, and complete or partial intestinal obstruction. I have made careful post-mortem examinations on several of these cases, and beyond extreme distension of the intestines have discovered nothing. Such a condition is known as "Paralytic ileus," and the name expresses probably all that is known as regards its pathology. After every abdominal section there is more or less intestinal distension, accompanied by irregular peristaltic contractions causing severe griping pains, and it is rare for a patient to be able unaided during the first two or three days to pass flatus or motion. If this amount of distension, which may be regarded as normal, should increase, the position of the intestines in the abdomen becomes more and more cramped, till in the final stages active mechanical obstruction is caused every few feet by acute bends in the intestinal tube. Hence in the end the difficulty is due in great part to a mechanical cause.

The Diagnosis

Cases of intestinal obstruction are divided clinically into two classes, the acute and the chronic; but classical text-book varieties will comparatively rarely be seen. The diagnosis is based upon certain symptoms, and in some cases, unfortunately not in all, on certain definite physical signs.

The Symptoms.—Acute obstruction makes its appearance suddenly. Pain occurs as the first symptom, and is very severe, sufficiently so in many instances to cause marked shock. The pain is accompanied by sickness, which is shortly followed by vomiting. The vomited matter consists, at first, of the contents of the stomach and clear fluid, then bile-stained fluid, and finally the vomit becomes fæcal in character. The depression is more marked the more acute the attack. Ordinary symptoms of shock are present; the patient is pale and chilly, has a feeble pulse and drawn face, and a faint voice. There is complete constipation; no wind can be passed, and hiccough may be a troublesome symptom. The abdomen in this stage is retracted, and kept fixed in the endeavour to relieve the pain. The most lasting symptoms are distressing thirst and vomiting.

Pain.—Pain may be local at first, and it is possible that the patient may be able by its help to point out the neighbourhood of the obstruction; later, as a rule, the pain becomes more ill-defined, and is apt to be referred to the neighbourhood of the umbilicus. Although the pain may never be absent, there are, as a rule, intermittent paroxysms during which it is markedly increased. The pain is not aggravated, but may be relieved by pressure.

Local Tenderness.—Tenderness is of greater importance than pain as a localising symptom. It is frequently to be found over the site of the obstruction, and occasionally the contracting gut may be felt to harden at this spot.

Vomiting.—Vomiting is frequent in the early stages of the illness, but in the later becomes less so, though it continues throughout, and in acute cases is one of the most important and troublesome symptoms. It depends to some extent on the nervous organisation of the patient, occurring more frequently in children and in women than in strong-willed men. Vomited matter, with fæcal odour, is the most characteristic sign of intestinal obstruction. The large quantity brought up, the obstinacy with which the vomiting persists, and the force with which the vomited matter is ejected, are also strongly suggestive signs. Fæcal vomiting is so rarely met with in other conditions that special importance is always attached to it. It occurs, however, in septic peritonitis, but even then it is probably due to mechanical obstruction caused by distension and kinking of the intestines. In two cases of malignant disease involving the stomach and transverse colon, and causing a fistulous communication between them, which have come under my observation, it was present.

The tongue is usually moist and foul in the early stages, and there is fearful thirst; later, the tongue becomes dry.

Constipation.—Constipation at first may not

be complete, because it is possible for the bowel below the seat of the obstruction to empty itself of matter contained in it, though this is very rare. Just before death a motion is not infrequently passed. Rarely, though it is important to bear the fact in mind, diarrhœa may be the most prominent symptom of obstruction. The most marked case of this sort that I have seen occurred in a woman on whom I operated for stricture of the ileo-cæcal valve. Every two weeks she had an attack of diarrhœa, attended by severe griping pains, with marked gurgling and peristaltic contractions and bad-smelling vomit. The lower ileum was enormously hypertrophied and dilated, and at the time of operation (an interval), though every effort had been made to clear her intestinal tract, with apparent success, by purgatives, the small intestine, though not distended, contained several pints of fæcal matter. It appeared to me probable that this patient's ileum was never able to empty itself thoroughly, and that the attacks of diarrhœa every fortnight were explained by the discharge of a large quantity of irritating matter, gradually accumulated there, into the colon. Spurious diarrhœa, with tenesmus, is a not uncommon symptom of stricture far down the colon.

Passage of Flatus.—Flatus is never passed except in cases of intussusception or partial obstruction, even though the bowel be relieved of solid matter. In intussusception, along with blood and mucus, a small quantity of flatus may pass during the attack of tenesmus.

Temperature. — Temperature is normal or subnormal, and this may be an important diagnostic point.

Swelling of the Abdomen.—Though in the early stage the abdomen is contracted to relieve pain, a little later swelling occurs, and this may increase rapidly and impede respiration. In cases of partial and chronic obstruction the hypertrophied and distended intestine may be seen and felt contracting and dilating through the abdominal wall. This sign is pathognomonic of intestinal obstruction. It may also be possible to locate the neighbourhood of the obstruction if it can be ascertained that the contraction ends always at some definite spot.

A tumour may be found in intussusception and in fæcal obstruction; also in obstruction due to foreign bodies, cancer, tubercle, matting of adhesions, internal hernia, tumours pressing on the gut; and sometimes the empty coils of intestine pressed down into the pelvis, can be felt from the rectum as an indefinite swelling.

Chronic obstruction begins gradually. It is often preceded by a period of loss of health and weight, and dyspepsia with constipation. The constipation is at first entirely relieved by a purge, but a characteristic history is one which tells of increasing rumblings of wind and pain after the administration of a purge. Later the patient may emit wind, though no stool passes. In cancer there is sometimes blood, spurious diarrhœa, and tenesmus. In chronic intussusception the same symptoms may be present. The stools, when the large intestine is diseased, may be altered in shape, and become tape-like or small worm-like in form. Frequently, on exposing the abdomen, contractions of the dilated and hypertrophied colon are visible. Vomiting occurs in the later stages, but fæcal vomiting is generally long delayed. The pain is intermittent, and is dependent upon forcible contractions of the intestine above the obstruction. The general symptoms are wanting at first. The end of the case is usually brought about by an acute attack supervening on the chronic course of the disease.

In a variety of acute abdominal lesions the symptoms are similar; for example, in peritonitis due to perforation of any of the viscera, in ovarian tumour with twisted pedicle, in ruptured or leaking ectopic gestation, in an attack of gall-stones or renal colic, in cholera, and in certain kidney cases; so that the first question to be answered on seeing such cases is:—

1. *Is it one of intestinal obstruction?*

The history is of value. If the attack be due to appendicitis there is frequently a history of previous ones, and it is rare for the perforative variety of appendicitis to start quite abruptly. If due to the perforation of a gastric ulcer there will probably be a history of previous stomach troubles. In ruptured or leaking ectopic gestation there may be the usual history of pregnancy, a missed menstrual period; if to an ovarian tumour, the tumour may have been observed before its pedicle became twisted. Perforating typhoid ulcer is preceded by illness of a febrile character. In kidney cases there may be albumin in, or total suppression of the urine. If the attack be due to gall-stones, a history pointing to attacks of hepatic colic, followed by jaundice, may be elicited.

The diagnosis from peritonitis is especially difficult. In peritonitis the pain is more rapidly generalised and more superficial; there is more abdominal tenderness; the abdomen swells more rapidly; the muscles become markedly rigid; pain and tenderness are increased by pressure, and the vomiting is less abundant and more rarely fæcal than in obstruction; the constipation is also less complete. In peritonitis the temperature is high at first, and later it may be lower than normal. In obstruction it is lower at first, and may be higher later when peritonitis develops. In peritonitis there may be signs of peritoneal effusion, and the abdomen becomes dull in all the most dependent parts. In obstruction intestinal movements may be visible.

The diagnosis, even if the greatest care be

taken, is not always possible during the earlier stages.

The case is likely to be one of intestinal obstruction if the patient, previously in good health, has been seized with sudden pain, which is much increased at times, if there is entire inability to pass flatus, and if vomiting is an urgent symptom.

The next proceeding is to make a complete examination. The abdomen should be freely exposed, the patient lying with shoulders raised in an easy posture, and in as good a light as possible. The hernial sites are to be first carefully examined, not only the ordinary ones, but the extraordinary as well. An absent testicle on one side would suggest a strangulated hernia, possibly hidden in the inguinal canal.

It is impossible to overrate the importance of a careful examination for hernia in the first place, for the most skilful practitioner, if he should forget this, will sooner or later commit an error fatal to his patient; indeed, I go further, and teach students that the first thing to be done in every case of urgent vomiting is to examine carefully the hernial sites, whether the patient considers it necessary or otherwise, and to believe if a hard lump is found there that this has *everything* to do with the condition until the contrary is proved by operation. The whole abdomen should then be systematically and carefully inspected, palpated, and percussed. A rectal examination must never, under any circumstances, be omitted, and in women, when possible, a vaginal examination should also be made.

2. *Where is the block?*

In acute obstruction the probabilities are that the block is in the small intestine, and in chronic obstruction that the block is in the large. The reason for this is that chronic obstruction is mainly due to stricture of the intestine, and the majority of strictures are malignant and in the large intestine. Though this general rule holds good in the majority of instances it has many exceptions. I have seen acute sudden obstruction produced by malignant strictures at the ileo-cæcal valve, at the hepatic and splenic flexures of the colon, and in the sigmoid flexure; and in one of the most chronic cases that has come under my notice the obstruction was in the middle of the jejunum, and due to adhesion and kinking of the bowel.

Pain localised by the patient may be of use as a guide to the site of the obstruction if the localisation is made quite early in the case. Pain, however, is a doubtful help, as it is mostly referred to the neighbourhood of the umbilicus. A tender spot is a much more certain guide, and becomes of great importance if a swelling or feeling of resistance is felt in its neighbourhood. In chronic obstruction the patient may feel pain which begins or ends at one spot, and flatus rumbling in his intestines may be felt by him to stop there. On inspecting the abdomen, in exceptional cases, moving intestinal coils may be seen, and their movements may be directed to one particular spot; and on palpation the peristaltic wave may be felt to be arrested there, producing a feeling of temporary firm tumour under the examining hand. This, in chronic cases, is a valuable guide as to the site of the obstruction.

A careful examination should be made in the early stage, because shortly the general swelling of the intestines which takes place is likely to obliterate any landmarks.

The shape of the abdomen is an aid in forming an opinion as to whether the obstruction is situated in the large or in the small intestines. When the small intestine is involved low down the abdomen becomes globular in form and prominent in the middle line, a similar distension to that caused by an ovarian tumour. When the large intestine is involved the abdomen is flattened and bulging at the sides, as in ascites, and may be enormously distended. A flattening at the left side, with enlargement on the right, points to an obstruction in the commencement of the transverse colon, the swelling on the right side being constituted by the distended cæcum and ascending colon.

If it can be made clear that the cæcum is distended the obstruction is certainly below this point. In some cases the distension of the cæcum is quite evident. It may be seen to swell up intermittently, to contract, and to harden, and peristaltic waves spread from it along the ascending colon towards the right costal margin. The abdominal swelling will also, in the early stages, be limited to the right half of the abdomen. There may be splashing on concussion of the cæcum. The pain and contraction are felt by the patient, and may be seen to begin in it.

A ladder-like arrangement of the distended intestine, seen in some chronic cases, is an aid to localisation, for this occurs only when the small intestine is involved. Very firm and painful contractions, in which the distended intestine becomes as hard as the contracting uterus, occur, so far as I have seen, only in the small intestine, for the colon and the stomach contract more slowly and more feebly than the small intestine.

Vomiting in the case of obstruction of the small intestine is more rebellious than in the large, and sooner becomes fæcal in character. In the case of the large intestine vomiting is usually a late symptom, and is rarely fæcal.

The general condition of the patient is more serious in small intestine cases. Depression and feebleness are more marked.

In cases of chronic obstruction of the small intestine anuria is a symptom of value, and is due to limited absorption of fluids and to their loss by vomiting.

Muscular cramps in the feet and legs are mostly met with in acute cases where the small intestine is affected.

The use of an enema I have frequently found to be a great help. If a small one only can be retained (say, less than one quart), this sign is in favour of the obstruction being in the large intestine.

An examination of the patient under chloroform may discover a tumour in one or other part of the abdomen. Distended intestinal coils felt per rectum point to obstruction in the small intestine.

As aids to diagnosis the passage of the long tube or a sound into the rectum are useless, and the introduction of a hand into the rectum is likely to convey very little information, and is a dangerous operation.

3. *What is the nature of the obstruction?*

If the case is one of acute obstruction it is probably due to acute intussusception or volvulus, to strangulation by a band or a ring, or to an internal hernia or gall-stone.

Intussusception.—Intussusception is the simplest form for diagnosis. It occurs most commonly in early youth. The attack is often preceded by a free intestinal evacuation. There is sudden pain—continuous at first, becoming, later, more intermittent. There is vomiting, which is not so severe or so persistent as in other acute cases; constipation, which is not likely to be complete, as it is usual to have small loose motions with muco-sanguinolent discharge and marked rectal tenesmus. The abdomen is seldom much swollen; indeed, it may be retracted, as relief is obtained by the passage of small quantities of gas per anum. Examination of the abdomen may discover at the ileo-cæcal junction, or in the course of the colon, a rounded sausage-shaped tumour, which hardens at times, or there may be merely marked local tenderness. Per rectum, it may be possible to feel the end of the intussusceptum, like a uterine cervix, or, if the tumour is too high for this, bimanual examination with one hand on the abdomen and a finger in the rectum may make it plain. A blood-stain on the examining finger may also give a clue. The swelling in the ileo-cæcal region may be mistaken for the swelling due to an abscess in connection with the appendix; but the history of the onset, the absence of intestinal symptoms characteristic of intussusception, the febrile course, and the tender, indefinite lump, as compared with the rounded and more readily palpable swelling of intussusception, serve as distinguishing features. In young children the diagnosis is occasionally very difficult. The typical symptoms are masked by what I can only describe as apathy. Except for vomiting there are no definite abdominal symptoms. The patients lie in any position in which they may be placed; with sunken features, looking very ill and apparently anæsthetised; they tolerate examination abdominally and rectally without complaint. In consequence of the relaxed and anæsthetic condition of the abdominal wall, the tumour is readily discovered.

Volvulus.—Volvulus occurs in elderly adults with a history of long-standing constipation. The pain and tenderness are most marked in the left iliac region, and there a vague swelling may be felt. Vomiting is not so frequent as in other forms of acute obstruction, probably because older persons are not so frequently sick as young ones are. There is absolute constipation, and frequently rectal tenesmus, but no tumour can be felt on rectal examination, and there is no bloody or mucous discharge. Abdominal distension appears early and rapidly increases. Later it is frequently most marked in the epigastrium.

Strangulation by bands, diverticula, rings, and internal hernia are often sudden in their onset, and produce very acute symptoms. A history of previous peritonitis suggests strangulation by a band or adhesions. In vigorous, previously healthy, young adults, Meckel's diverticulum should be remembered. In subjects of Glénard's disease with gastroptosis, movable kidneys, etc., retro-peritoneal hernia may be suspected. This may form a palpable tumour on the left side of the abdomen. Patients with strangulated diaphragmatic hernia are frequently unable to lie on the left side in consequence of difficulty of breathing, and complain of trouble in their chest. The upper part of the abdomen in these cases may look hollow, and percussion and auscultation yield signs of stomach and intestine in the thorax. In gall-stone cases there is sometimes a previous history of hepatic colic or of peritonitis in the neighbourhood of the liver. Stout old women are the favourite subjects for it. In them the obstruction symptoms may commence acutely, then subside into those of incomplete obstruction, with passage of more or less flatus and partial relief. Not infrequently strictures, malignant or simple, first draw attention to themselves by producing an acute attack of intestinal obstruction, though it is usual on close inquiry to get a history of previous trouble.

In the *chronic form of intestinal obstruction* tumours external to the bowel should be first excluded as the cause. Uterine and ovarian tumours are perhaps the most common. Then chronic peritonitis must be eliminated, and this is difficult when the general abdominal swelling is great. The diagnosis is mostly to be made between the following causes of obstruction:—Fæcal masses, strictures, adhesions, chronic intussusception (for rectal cases *see* "Rectum").

Fæcal masses are mostly found in feeble old persons, hysterical women, and lunatics. The history is one of long-standing constipation and of gradually increasing abdominal swelling;

there are borborygmi and attacks of colicky pain. Vomiting does not occur until late. A series of masses, which are not tender, and which are hard and movable, may be felt in the course of the colon. There is frequently fœtid diarrhœa from the irritation of the mucous membrane of the intestine by hard putrid collections. Tenesmus is marked when the hard lumps pass into the rectum. Rectal examination may reveal scybala there, and help the diagnosis. Purgatives and enemata may not for some time remove the swellings and relieve the symptoms. This form of obstruction is unfortunately regarded as common, and it must be borne in mind that it is easier to mistake malignant masses for hard fæces than the reverse.

Cancer and stricture are usually confounded because the form which cancer takes is frequently annular, and the intestine is in reality strictured by it. In a patient over forty years of age cancer should be suspected. A tumour at the ileo-cæcal valve, or the hepatic or splenic flexures, or in the sigmoid, may be discovered. In women a vaginal examination should seldom be omitted. The sigmoid flexure with a malignant growth in it, from its weight will sink into the lower part of the pelvis, and may be felt there from the vagina, lying at the bottom of the pouch of Douglas. Attention to this point has more than once enabled me to make a definite diagnosis of malignant stricture of the sigmoid flexure. The tumour, if rounded, hard, and nodular, as it frequently is, closely resembles an enlarged and prolapsed ovary. In men a similar tumour of the sigmoid may fall into the lower part of the recto-vesical pouch, and can be recognised by a finger in the rectum, most easily when the patient is standing upright. As a rule constipation is the first symptom. It yields to purgatives in the early stages, but gradually becomes more obstinate, when it may last for some days, and be finally relieved by a purgative and enemata. A very suggestive history is that purgatives caused so much intestinal commotion as to produce rumbling and fluid-dropping sounds loud enough to be heard by any one in close proximity to the patient.

Pain in all chronic cases is characterised by its severe paroxysmal character, and is attended by visible intestinal movements along with the rumblings. After the pain has gone the patient may feel well for weeks or months, but as time goes on the attacks become more frequent and more severe. Towards the end it is usual to have sickness and vomiting. In malignant cases sometimes there is blood found in the stools, which are also loose and fœtid. If this occurs late in the history of the case it means ulceration of the growth, and possibly is attended by greater freedom in the passage of the intestinal contents following this destructive process.

In simple stricture a history leading to the diagnosis of tuberculosis, syphilis, dysentery, or typhoid may be obtained, or that the symptoms were preceded by a violent contusion of the abdomen, or by a strangulated hernia. There is a history of attacks of pain usually occurring after food, and frequently attended by vomiting. Associated with these are rumblings of wind and attacks of constipation, or alternately diarrhœa and constipation. The paroxysms become more frequent, vomiting and constipation more severe, and abdominal distension with vigorous peristaltic movements develop.

If a tumour is felt at the ileo-cæcal valve it is difficult to say whether it is tuberculous or cancerous, or due to chronic intussusception. A tuberculous tumour, ill-defined and caky, generally occurs in young subjects with the pallor and weakness that are suggestive of the constitutional feebleness common in tuberculous subjects. It is accompanied or preceded by diarrhœa, and there is frequently a nocturnal rise of temperature, with night sweats. Other evidence of tuberculous taint may be available —the scars of old suppurating neck glands or changes in one or other lung, or the scars of some tuberculous skin, bone, or joint affection.

The cancerous tumour, hard and nodular at the ileo-cæcal valve, occurs in elderly subjects, and is preceded and accompanied by constipation, because of the obstruction which the growth located at the valve causes to the passage of intestinal contents. Occasionally blood will be passed per rectum. The patient may have the cachectic appearance associated in the minds of so many with cancer, but it is quite as common to find the look of robust health.

In chronic intussusception there is intermittent pain; during the intervals the patient may be quite easy. There are muco-sanguinolent motions and tenesmus, and a cylindrical tumour softer than the cancerous or tuberculous one, and perhaps hard at one time, soft at another, and altering in position. Vomiting is not a marked symptom. The bowels are regular, or there may be diarrhœa. There is no general swelling of the abdomen. The patient gradually gets thinner and weaker, and this it is which frequently suggests that the disease is cancer.

Strictures both simple and malignant may become occasionally blocked by hard fæces or foreign bodies, and the patient then develops acute symptoms.

Chronic obstruction, due to adhesions, is suggested by a previous history of peritonitis, possibly tuberculous, or consequent upon pelvic disease, or following an appendicitis. In such cases the symptoms are apt in the early stages to be badly characterised; they develop slowly, and run a course which shows the difficulty of drawing the artificial distinction which we have attempted to do between acute and chronic obstruction. At either end the distinctions are

clear enough, but in the great majority of cases of intestinal obstruction it is impossible to say that the acute will remain so till the end, or that the chronic will escape an acute termination, and in these cases the commencement may be acute, and after a day or two of serious illness the symptoms become more chronic, or partially relieved by the passage of flatus.

A distended abdomen with paroxysmal pain and fœtid diarrhœa is seldom met with except in chronic obstruction of the colon, and diarrhœa is indeed so common in cancer of the colon that it is imperative to examine the rectum in every case of obstinate diarrhœa in the adult.

Prognosis

The difficulties of correct diagnosis and, consequent thereon, of exact prognosis have increased the uncertainties of treatment. Before abdominal surgery reached its present position, if there were a fair chance for the case to recover without operation, there was every excuse to delay operative treatment, which had so high a mortality in intestinal obstruction cases. This excuse no longer exists, and it is fair to say now that the mortality after operation is more due to delay, or to diseased conditions which could not have been relieved at an earlier stage, than to the operation itself.

Acute Intestinal Obstruction.—In acute cases the mortality is very high. Strangulation by bands or rings or internal herniæ will have as high a mortality as cases of strangulated hernia would if treated without operation. It is possible for either to get well when apparently in the most desperate condition, but such cases are so rare that they cannot be allowed to count in making the prognosis.

Post-mortem examination made by me of a stout and apparently healthy woman, who died suddenly twelve hours after the onset of obstruction symptoms, showed that death was due to a small knuckle of the ileum being strangulated through a hole in the mesentery. On another occasion in a vigorous young blacksmith, who lived for three weeks after the onset of acute obstruction, death was found to be caused by a band crossing and constricting the end of his ileum.

The great majority of cases die during the first week, and seldom live for more than three days after fæcal vomiting has commenced. Death in these cases is frequently brought about by changes in the intestine above the obstruction. Interference with the circulation, due to distension, first destroys the lining epithelium of the mucous membrane, and allows of the absorption of septic matter into the circulation. Sudden death may take place from heart failure during this stage, or if the patient live longer, death arises from a combination of septicæmia and exhaustion due to pain, loss of sleep and starvation. Later, septic absorption with peritonitis caused by the escape of bacilli through the damaged intestinal wall, or perforation of distension ulcers, may cause death. The latter is rare. A more usual termination is by peritonitis due to the perforation of ulcers in the neighbourhood of the seat of obstruction.

Strangulation by Bands or Internal Hernia.—The most acute cases so closely resemble strangulated hernia that the prognosis in both may be said to be the same without operation.

Intussusception. — In acute cases recovery without surgical intervention is more common than in other varieties of intestinal obstruction.

In children under one year of age the prognosis is worst. They often die in a few hours with all the symptoms of profound shock; in older children and adults death usually occurs in the second and third weeks of the illness. An adult female under my care, with all the symptoms of intussusception, with fæcal vomiting of three days' duration, and a tumour in the position of the ascending colon, suddenly recovered with the disappearance of the tumour, and there is in recorded cases considerable justification for the belief that spontaneous reduction may occur. This, however, must be regarded as an unusual result. The common cause of spontaneous cure is gangrene of the intussuscepted bowel. Gangrene may affect the whole or only part of the invaginated bowel. In a child I saw after it had been ill for a week with symptoms of intussusception, there were several inches of gangrenous intestine with the ileo-cæcal valve at its extremity hanging out of the anus.

The obstruction, as would be expected, is most complete and sudden in acute cases, as in them the vessels of the involved mesentery are at once constricted, and large portions of the gangrenous but intact intestine may be passed per anum. In more chronic cases destruction may be less rapid and more partial. Instead of large areas of gangrene, smaller sloughs of the invaginated intestine may be voided, which may be difficult to recognise as portions of the intestine when passed per anum.

In adults the possibility of polypi as a cause of intussusception has to be remembered, and in elderly persons the still more frequent association with cancer must be borne in mind in making the prognosis.

In the very young, spontaneous elimination by gangrene is almost inevitably fatal.

In youth the prognosis is best; after twenty it becomes steadily worse.

Collected statistics show that out of one hundred cases of intussusception fifteen will recover after spontaneous elimination by gangrene of the intussuscepted bowel. It seems probable, however, that these statistics give too favourable a record for spontaneous cure by gangrene, as recoveries are more likely to be reported in consequence of their interest and rarity than the more usual fatal results.

Spontaneous elimination is usually attended by foul stools containing some blood and masses or shreds of gangrenous intestine, and is followed by diarrhœa. It can only end in cure when the ensheathing and entering layers of gut are firmly glued together by adhesions. The most common cause of a fatal termination is perforation at the neck of the intussusception, and from imperfect union by adhesion of the opposed surfaces. This latter leads to a general peritonitis, or to local peritonitis with abscess, and though the illness in the latter case is usually more prolonged, the ultimate result is the same. Other patients die because the surface at which separation has occurred does not heal, and remaining ulcerated causes diarrhœa, or the ulcer eventually perforates, or causes fatal hæmorrhage from the opening of a mesenteric vessel, which may also occur during the separation of the dead part from the living, or the ulcer produces the different forms of septic disease by absorption of septic products from the raw surface, or as a remote result stricture of the gut may follow the cicatrisation of the ulcer.

Volvulus of the Sigmoid Flexure.—The rapidity with which distension of the bowel occurs and adhesions follow at its neck would make it very unlikely that spontaneous recovery would occur in this condition. Death has occurred a few hours after the onset of symptoms, apparently from shock. Septic absorption from the damaged intestine is the most frequent cause of death, and next to this comes peritonitis. Sudden death from heart failure is even more common in this form of intestinal obstruction than in others, and death from one or other of these causes is almost inevitable during the first week unless the condition can be remedied by operation.

Gall-stones.—About 50 per cent of cases with definite obstruction symptoms due to this cause die during the first ten days. The causes of death are chiefly changes in the intestine above the obstruction leading to septic absorption and peritonitis. Patients who do recover have to pass through a more or less severe illness, followed by evacuation of the gall-stone.

The prognosis of all acute cases of intestinal obstruction taken together is probably a recovery rate of about 15 per cent.

Chronic Intestinal Obstruction

Stricture of the Small Intestine.—In simple stricture the symptoms may be of very long standing—several years, in cases where great attention has been paid to details in matter of diet, and to the regulation of the evacuations. Such a chronic course is only made possible by compensatory hypertrophy of the intestinal walls above the obstruction. In some instances the intestinal walls may be as thick and as strong as those of the stomach. In one of my cases the small bowel reached the size of an ordinary forearm, and served as a receptacle for immense quantities of semi-digested food.

In cases of malignant stricture affecting the small intestine, it is rare for any relief to be possible except through palliative operation.

Spontaneous relief in both simple and malignant cases may occur from ulceration and abscess above the obstruction, if the abscess opens externally and forms a fæcal fistula; it may also, though more rarely, occur from the formation of an anastomosis between the intestine above the stricture and that below it by a fortunate combination of ulceration, adhesion, and perforation. Except for these extraordinary and rare chances the course of intestinal stricture is to a fatal termination. Death is generally brought about by the supervention of an acute attack produced by some object, such as a solid fæcal mass, or a grape-skin or fish-bone, or a cherry-stone, completely obstructing the small lumen of the strictured part of the bowel, or by a kink at the narrow portion. It may also result from malnutrition, caused by interference with the due performance of the digestive and absorptive functions, or it may be brought about by peritonitis, usually due to perforation.

Stricture of the colon always ends in death, but life may be considerably prolonged by a suitable diet and laxative medicines. Many cases have been recorded in which persons with stricture of the colon lived for several weeks without any intestinal evacuation.

The average duration of symptoms is under one year. The average duration of life in cancer of the colon is fifteen months, allowing that palliative operation (colotomy) has been performed for the relief of the obstruction.

The causes of death are the supervention of an acute attack, general peritonitis due to ulceration with perforation above the stricture, or local peritonitis followed by abscess and septicæmia, or gangrene of the stricture from blocking of its vessels of supply by growth in the mesentery. Gangrene causes perforation, and death is brought about by peritonitis.

Spontaneous relief may follow ulceration and the formation of an artificial anus above the strictured part. This may open externally or into the bladder, or into the vagina of the female. In a more rare case upon which I operated for obstruction due to malignant stricture of the sigmoid, gangrene of the entire cæcum and the greater part of the ascending colon from extreme distension was found on opening the abdomen, and the patient was relieved for some weeks by the natural colotomy resulting from the separation of his gangrenous cæcum and ascending colon. Relief may also follow the formation of a natural anastomosis of

the intestine above the stricture with that below.

After colotomy in cancer cases death arises most commonly from cancerous involvement of the peritoneum or of the liver; less frequently, from secondary growths in other organs; more rarely it follows sloughing of the diseased part due to blocked mesenteric vessels, followed by localised abscess or general peritonitis.

In chronic intussusception the prognosis is grave. Spontaneous elimination is very rare. After a month of illness, with no sign of spontaneous elimination, death is almost inevitable without surgical operation. The fatal termination generally arises from malnutrition, due to interference with digestion and the absorption of food, or from the supervention of an acute attack, or from perforative peritonitis.

In fæcal accumulation death is very rare, except in lunatics, because the treatment usually adopted proves effective. Death may occur from malnutrition, or from perforation, or from rupture of the distended intestine, the cæcum or the sigmoid flexure being the part to give way.

Treatment

The treatment of intestinal obstruction has been made uncertain by difficulties in diagnosis and prognosis, and because both are vague the choice of treatment has been wanting in decision. It must at once be admitted, for every one who has had experience in this disease knows it, that the most unpromising case, in which the symptoms are so marked that a diagnosis of obstruction by a mechanical obstacle is indisputable, may recover spontaneously or under medical treatment.

We also know, however, that treatment by the most approved medical methods, and carried out completely from the onset, results in an appalling mortality; and the same may be said of operations performed as a last resource. Available statistics are useless as an aid in our endeavour to formulate the best methods of treatment. There are no figures to show what is the mortality of operations performed during the first thirty-six hours of the illness, and this should be the extreme time limit if operation is to have the best chance. My opinion, based upon the pathological causes of intestinal obstruction and the general results of abdominal surgery, is that the recovery rate would reach 70 per cent at once if the rule to operate early were universally adopted. In support of this opinion, it may be well to state that of 8 acute cases of intestinal obstruction operated upon during the past year of less than fifty-three hours' duration, in which I have been concerned, 6 have recovered and 2 have died.

Before considering in detail the treatment to be adopted in a case of acute obstruction, I cannot too urgently impress the need for an exhaustive examination, and the formation of the best possible diagnosis, at any rate as to the dangerous character or otherwise of the illness before treatment is commenced.

The most urgent symptom, and the one which always demands the earliest possible relief, is pain. Opium in one or other of its forms is the only drug known which can be relied upon to do this safely, effectually, and speedily. Considering the condition of the stomach and upper intestine, it would appear to be unwise to administer opium by the mouth, as it may be ejected in whole or in part, or, if retained, may fail to become absorbed till, perhaps, the obstruction is relieved, when its effect is not desired, and when indeed a poisonous quantity may find its way into the circulation. The rectum is a better channel for its administration, when it can be given as a suppository or in a small enema; but the best, because the surest and quickest, method is to give a dose of morphia hypodermically. Too large a quantity is to be avoided as hurtful, and one too small, as it will fail to give relief, and may require repetition. A third of a grain in an adult may be generally said to be the proper dose. Repeated small doses of opium are to be condemned in all acute abdominal cases. Administered in frequent small quantities, the drug masks all symptoms and leads to a false sense of security; it paralyses the intestines; and it produces a hyperæsthetic condition of the patient very unfavourable to recovery. A reduction of the accustomed dose leads to a miserable condition of physical and mental collapse, and patients under its influence are predisposed to die from shock after an operation. Though it is true that the rest, intestinal and general, brought about by the use of opium may result in a marvellous improvement in the general condition, I doubt whether the curative influence ascribed to it by generations of authorities has any foundation in fact. If a loop of intestine has been ensnared by a band, it appears to be as reasonable to suppose that, by violent peristaltic efforts, it might again escape as that its release was to be aided by keeping it quietly at rest.

It may have been obvious before the administration of opium that the patient was dangerously and acutely ill, and in all such cases the abdomen should be opened without waiting to see what the result of the opiate may be. Others less certainly ill should be seen at short intervals and closely watched. In ordinary cases a large, hot linseed-meal poultice applied so as to cover the entire abdomen, and left on for an hour, has a soothing effect, and the heat of it diminishes shock, from which the patient is probably suffering. If it be removed at the end of an hour, and, after sponging the skin with hot water and drying it, cotton wool be wound round the body, the weight of the poultice can scarcely be seriously objected to. During the first hours nothing whatever should

be swallowed except a few sips of water as hot as can be tolerated, and this should be limited to as small a quantity as the patient's craving for drink will allow. At the end of eight hours, as the effect of the narcotic begins to wear off, if the symptoms return operation should be advised. When the symptoms, then, are not sufficiently definite, and the diagnosis still remains doubtful, my practice is to order calomel in one-grain doses every hour until five grains have been taken, and after the fifth dose to administer a turpentine enema (one ounce of turpentine in a tumblerful of barley-water), followed by one pint of soap and water. If the case is one of obstruction, the symptoms, which are likely to have disappeared soon after the opiate has been given, will probably all be reproduced by this treatment, and the best chance—that given by early operation—is secured. If the case is not one of obstruction this treatment is likely to be the best available, for the cases most closely simulating acute intestinal obstruction are those of so-called colic which occur in young men from eighteen to twenty-five, and are due to chronic constipation and partial retention of fæces.

If the case is not seen until the abdomen has become distended, and the symptoms have all been rendered less aggressive by repeated doses of opium, the question of operation has lost much of its urgency. The condition of the patient may be very serious, but operation may not afford the best chance of recovery. I have seen such seriously ill persons recover under medical measures who, so far as I could judge, would have died had they been subjected to the shock of operation.

The most favourable cases for recovery, without operation, and they only should be left alone, are those in which the obstruction is not absolutely complete.

A little flatus may have been passed, and occasionally peristaltic movements may be perceived in the distended intestines.

The contents of the stomach are expelled with vigour, and vomiting gives relief; quantities of nourishment can be retained and absorbed.

The distension is not so great as to produce difficulty in breathing or marked tension of the abdominal walls, and is not increasing.

The pulse is not growing more rapid or its vigour decreasing.

In the less acute cases, when vomiting has ceased, small quantities of liquid nourishment, half milk and half barley-water, probably being the best, may be tried; and if this is tolerated by the stomach, additions may be carefully and gradually made as the conditions allow.

The three most material palliative aids to recovery are:—Washing out the stomach, large nutrient enemata, and repeated small doses of opium. Persons differ much in their tolerance of stomach lavage, and it must be allowed that occasionally persistence with it may do more harm than good. The tube used should be a red rubber one, which the patient is assisted to swallow, and attached to it should be a glass T-piece to act on the principle of Sprengel's pump, as neither the efforts of the distended stomach nor the aid of ordinary syphon action may be sufficient to empty the stomach of all its contents. The washing should be done with water at a temperature of 102° F., and continued until the wash water returns clear. If this operation is to be of service the patient must feel relieved by it, and will consider the gain greater than the discomfort to which he has been subjected, and this should be the test as to its usefulness and as to the desirability of repeating the proceeding. If the use of the tube is found to be too disagreeable, the best substitute is to allow the patient an occasional hot drink (a pint of very weak hot tea without sugar or milk is nearly always to his taste). This is soon ejected, bringing with it a quantity of the foul stomach contents. Very soon after the stomach is emptied it commences to refill from the discharge of the distended intestine into it, and the washing requires to be repeated every four hours.

Inability to bear the tube should be regarded as indicating the advisability of early operation.

No food should be allowed to enter the stomach so long as sickness is a prominent symptom. Food and fluids introduced into the stomach only add to the sufferings of the patient, and often diminish what small chance of recovery is left to him. To relieve the intense thirst sips of hot water may be frequently given, and the mouth may be sluiced round with cold water as often as desired by the patient. Ice gives only temporary relief, and does permanent harm. It makes the mouth sore and the tongue dry; it fills the stomach with cold water, and increases flatulent distension.

All nutriment should be administered by the colon. The best method of doing this is to give slowly through a rectal tube and raised funnel one pint of a mixture of fatty beef-tea and milk in equal parts, with one teaspoonful of salt and half an ounce of whisky at a temperature of 100° F. every two or three hours. The first two or three pints are frequently retained when given in this way, whereas the intermittent pumping action of the ordinary enema-syringe commonly used for this purpose stimulates the bowel to expel the injected fluid. When the enemata begin to come away, it is, of course, a favourable omen if some flatus is passed at the same time. Diminution of the abdominal distension, as shown by careful measurement round the umbilicus, is also a favourable sign. If the enemata are retained for twenty-four hours it is necessary to administer a quart of salt water to clear out the offensive débris, as in all cases where food is

given by the bowel. From the use of nutrient suppositories little good need be expected, and if the enemata cannot be retained their expulsion is a strong indication for early operation. In connection with enemata, it may be mentioned that their use is strongly recommended by nearly all authorities in the treatment of intussusception. It is agreed that they have no value in the most acute cases or in cases involving the small intestine. It is also agreed that there is a certain amount of danger in their use. Before giving the enema, if this is decided upon, an anæsthetic must be administered to complete relaxation. Hot salt water, a drachm to a pint, is the least irritating injection, and as much of it as can be retained should be introduced. The enema funnel should not be raised more than three feet in the case of children, who may be held up by the feet during the administration. It is usually impossible to say when this operation is completed, even when a tumour can be distinctly felt and disappears, that reduction has been accomplished, and valuable time may be lost in waiting to see if the symptoms return. It is usually safe to prophesy that they will do so, and, except in the more chronic cases, in which a short period may be allowed to be wasted with comparative impunity, operation ought to be done as early as diagnosis permits.

Repeated small doses of morphia in the favourable cases previously mentioned are often of great service—10 ♏ of liq. morphia hydrochlor. in one drachm of peppermint water, to be given by the mouth, when the patient is restless or in pain, secures bodily and mental rest, and has a stimulating effect on both mind and body.

If nothing short of narcotism brings relief, operation is urgently indicated.

In addition to the use of stomach lavage, nutrient and stimulating enemata, and morphia, which are the most important means, there are smaller details requiring attention. A hot, moist poultice, large enough to cover the whole abdomen, kept on for an hour and repeated at intervals of eight hours, and replaced by warm cotton wool, is useful. A suitable bed and a good nurse are great comforts, and are important aids to recovery.

Operation in Acute Intestinal Obstruction.—In cases of acute intestinal obstruction the sooner operation can be performed the better the prognosis. It is, indeed, now amongst surgeons an established rule that there should be no unnecessary delay in opening the abdomen for any cause producing sudden alarming symptoms, and the sooner this rule is applied to cases of intestinal obstruction the better. That the dangers of delay far outweigh the dangers of operation, serious though these may be, is the most important point to bear in mind in connection with the treatment of intestinal obstruction. During the first forty-eight hours it is usually possible to perform an operation which allows the cause of the obstruction to be dealt with, and which offers good prospects of complete success.

Preparation of the Patient for Operation.—As soon as excruciating pain has been relieved by morphia, and the symptoms of collapse have subsided, the skin of the patient is prepared as for an abdominal section. It is of the greatest importance to have a warm room for the operation, and by hot bottles and warm clothing to prevent chilling of the patient on the table. The arms and legs should be swathed and bandaged. Children especially require exceptional trouble to be taken in this respect.

Any special instruments, such as intestinal clamps, Murphy's buttons, Paul's tube, should be ready for use, and everything should be prepared before an anæsthetic is administered.

Chloroform in the hands of a skilled administrator is the best anæsthetic for general use. Before its administration the patient's stomach should be washed out, to avoid the risk of regurgitating septic fluid being drawn into the lungs.

Abdominal palpation should be repeated under the anæsthetic, as a tumour which has previously escaped observation may now be discovered.

The incision is commenced one inch above the umbilicus, which is included in an ellipse, and prolonged to two inches below it. The umbilicus is then excised. Excision of the umbilicus removes that portion of the abdominal wall which is most difficult to disinfect. It allows of safe extension of the wound upwards and downwards, avoids the chance of damage to the bladder, and renders access to the abdominal cavity through the umbilical ring easier and safer than any other situation. The incision is then continued through the linea alba upwards and downwards from the umbilical ring, and the peritoneum is drawn forward between catch forceps away from the underlying viscera, and is opened. *The first object of the operation*—to confirm the diagnosis—will now be realised, for from the earliest stage the intestine above the obstruction is observed to be distended and increased in vascularity, and inflammatory peritoneal exudates are absent. The intestines are carefully kept inside of the abdominal cavity by a warm flat sponge or sterile swab, and a careful search is made in the neighbourhood of the wound, which should be opened by retractors. If nothing be seen there, two fingers should be gently passed in for exploratory purposes, and the most vascular and distended coils of intestine, which, it will be remembered, are above the obstruction, and which may be recognised as belonging to either the large or the small intestine, are gently pushed aside. *The second object of the operation* has now been attained, and

the obstruction localised in the small or the large intestine. Supposing the small intestine to be affected, and a pale and contracted loop, which points certainly to bowel below the obstruction, be found, it should be drawn forward and followed inch by inch until it guides to the seat of obstruction or to its termination in the ileum. In the latter case a journey will have to be made in the opposite direction, and this leads to the obstruction. Each portion of the bowel is carefully returned to the abdomen or protected by hot towels as it is passed through the fingers. The third point to be attended to in the operation, of finding the obstruction with as little disturbance or handling of the distended and friable intestine as possible, has now been achieved.

If the obstruction is not readily discovered in this way the abdominal incision should be prolonged to just above the pubis, taking care to see and to avoid the bladder, and the exposed intestines are to be covered with aseptic towels wrung out of hot normal saline solution. Any uncovered portion of intestine should be kept hot and moist by an assistant steadily dripping hot saline solution (ʒj. to 1 pint) over it and the surrounding coils and protecting towels from a jug during the whole time of exposure. Commencing on the right side, the condition of the cæcum, dilated if the obstruction is below it, will tell whether the obstruction be in the large or in the small intestine. If it is in the small intestine the contracted end of the ileum entering the cæcum may be followed up to its junction with the distended bowel above, and so discover the site of obstruction.

If the obstruction be localised in the large intestine, recognised by its longitudinal bands, the sigmoid flexure is first drawn up for examination, and if this be vascular and distended the obstruction will be found towards its lower end. If not, the transverse colon should be brought forward by drawing on the omentum, on the under surface of which this portion of bowel will be found; finally, the ascending colon and cæcum are examined.

As a last resource, by an extension of the abdominal incision to the ensiform cartilage and evisceration, any obstruction can certainly be found.

Evisceration adds greatly to the gravity of the operation, and should only be undertaken in vigorous subjects not reduced by long illness. It is to be preferred to rough handling of the distended and friable intestine, but, as a rule, the best proceeding at this stage is to draw out and to open a coil of tense intestine, for in any case the distended intestine must not be replaced in the abdomen. The most tense intestinal coil visible should be drawn forward well out of the abdomen, the opening into which should be isolated with aseptic gauze packs. When the extruded coil is thoroughly shut off from contact with the wound, it is opened by a longitudinal incision about half an inch long through the wall opposite to its mesentery, and the intestine is allowed to empty itself. If it does so without help, pumping out its contents vigorously, and the general condition of the patient is fair, after temporarily closing the intestinal opening with clamp forceps, a further search for the cause of the obstruction should be made, and this is rendered more easy by the diminished abdominal tension. If the obstruction is now discovered, and can be relieved, after careful suture and cleansing of the intestinal wound, the abdomen may be entirely closed. If, however, the intestine, though obviously distended and tense, refuses to empty itself without aid, and if, after a pinch between the finger and thumb, no peristaltic movement is seen in it, enterostomy offers the best chance of recovery, for if the paretic intestine is subjected to further manipulation neither it nor the patient is likely to recover.

If the obstruction is found to be an intussusception, and if it is acute, and the intussusception is swollen and firmly strangulated, and there are no adhesions, the surgeon's first duty is to lessen the œdematous swelling of the intussusceptum which is the chief barrier to reduction. The tumour is to be drawn forwards, wrapped in a large, warm, flat aseptic sponge, and gently but firmly squeezed by gripping it in the hand. The pressure must be continued till the swelling has subsided, then an attempt should be made to reduce the intussusception. This is done best by a combination of kneading from below and gentle traction from above, most reliance being placed on pushing from below, as it is easy to tear the intestine by drawing on it too freely. Adhesions may prevent reduction. These can be separated by a blunt director introduced between the ensheathing layer and the intussuscepted portion of bowel. If it is not possible to effect reduction, and the intussusception is of limited size, the best course is to excise the whole of the involved intestine. The abdominal cavity all round the intussusception is packed off with aseptic gauze, and the portion of intestine to be excised is completely isolated by this means. Two pairs of clamp forceps are fixed above the involved portion of intestine and two below. The intestine is divided between the two clamps above and below, and the intussusception is isolated. Then by gradually snipping through the mesentery with scissors, each vessel being secured before division if large, and caught as divided if small, the mass is separated. After ligature of the clamped mesenteric vessels and the arrest of all bleeding, the treatment of the divided intestine has to be decided upon. This depends upon the condition of the patient. If it is good, the continuity of the intestinal canal must be restored by an end-to-end or lateral

anastomosis, of which I prefer the latter. If the condition of the patient does not allow of this, the involved portion of intestine must be drawn out and packed round with gauze in such a manner as to thoroughly isolate it and block the abdominal wound; then the bowel is to be opened above the intussusception and slit up through the length of it to relieve the strangulation and permit of fæces passing through the artificial anus. This operation, if the patient recovers, requires a second one at a later date to restore the continuity of the canal, and this is a disadvantage, but the present safety of the patient must be the first consideration.

Volvulus.—After the abdomen is opened, the marked distension and vascular disturbance of the sigmoid flexure, easily recognised by its longitudinal bands and appendices epiploicæ, which become apparent, makes the diagnosis clear. An attempt should be made to guide a tube introduced through the anus into the distended bowel for the purpose of emptying it. If this cannot be done the patient should be turned over on to his side, and the wound, being protected by gauze, the presenting loop of sigmoid should be incised longitudinally and drawn forward by a pair of hæmostatic forceps fixed on either side of the incision. As the contents escape, the bowel is to be further drawn out from the wound by the forceps. When empty of its contents the bowel is washed, wiped dry, and the opening in it is closed with clamp forceps, the whole being covered with iodoform gauze. An examination of the empty sigmoid loop is then undertaken and the twist undone. The hole, if it has been necessary to make one in the bowel, is next closed with a continuous suture of catgut through all its coats, with the object of making it watertight and arresting all bleeding. This first row is covered in by a second row of fine silk Lembert sutures, and the bowel is cleansed and returned. Before closing the abdomen the centre of the loop should be sutured to the parietal peritoneum to prevent a relapse.

Bands.—As these mostly contain a blood-vessel, and are sometimes a hollow prolongation of Meckel's diverticulum, it is a wise precaution before dividing them to apply two clamps and cut between, subsequently ligaturing each end. The portion of bowel nipped by the band should be examined before letting it drop back into the abdomen. I lost a patient on one occasion by perforation of the intestine at the ulcerated site of a narrow band on the tenth day after operation. If the ring of the intestine does not recover its normal appearance after the application of a hot sponge, it should be doubled in by drawing the intestine above and below over it, fixation being secured by a few fine silk Lembert sutures. If then it should slough or ulcerate, it will do so safely into the lumen of the gut.

Internal Herniæ.—If a loop of the bowel ensnared cannot be withdrawn by the opening through which it has passed, without enlargement, the frequency with which a blood-vessel surrounds this opening has to be borne in mind. If possible, the safest method is to enlarge the aperture by stretching with sinus forceps passed through it guided by a director. The same precaution as to examining the nipped intestine is necessary in this case as in that of a band.

Cases of hernia into the duodeno-jejunal fossa have been recorded in which no portion of the small intestine was visible on opening the abdomen, the whole of the jejunum and ileum having passed into a pouch, of which this was the neck. A knowledge of this fact will make a recognition of the condition to be dealt with and its treatment easier.

Gall-stones.—A gall-stone large enough to cause obstruction will probably be readily recognised by intra-abdominal palpation. The portion of intestine containing it should be drawn forward and examined. If the stone is lodged close to the ileo-cæcal valve, a gentle attempt should be made to push it on into the colon, where it may be left to pass on by natural efforts. If this cannot be done, or if the stone is lodged in the jejunum, or higher in the ileum, it should be squeezed upwards for some inches into an unirritated piece of intestine; and after this has been drawn out of the abdomen, the intestinal coats over the stone should be incised longitudinally at the convexity of the intestine, emptied of the stone and other contents, and sutured in the manner previously described, and returned with the precautions for avoiding sepsis already mentioned.

Stricture of the Small Intestine.—If the stricture is simple and annular it is readily and safely dealt with by enteroplasty. In this operation the intestine is opened three-quarters of an inch above the stricture on its convex side, and a guide is passed on through the stricture, which is divided in the same line by an incision extending three-quarters of an inch below the stricture. The longitudinal wound is cleansed and sutured transversely with an internal row of catgut sutures passing through all the coats, and an external row of silk Lembert sutures burying it. The same treatment is also occasionally the best palliative for annular malignant stricture of the small intestine which is not removable as such strictures usually are, but as a rule in these cases an anastomosis above and below is the most satisfactory method of treatment. More complicated strictures, as for example those associated with active tuberculous disease, are best treated by excision of the diseased and strictured portion of the intestine, or by making an anastomosis above and below the stricture. If the condition of the patient does not allow of this, enterostomy

above the stricture must be performed as a palliative measure.

Stricture of the Large Intestine.—If the patient is vigorous enough to allow of exploration, the operation should be conducted with two objects in view—the remote one of a radical cure, and the immediate one of giving relief. This exploration, which should be accomplished in a few minutes, should demonstrate for future guidance the exact situation of the disease; its character, simple or malignant; if malignant, whether fixed or movable; and the presence or otherwise of enlarged glands or secondary deposits in the liver and elsewhere.

It is seldom wise to attempt a radical cure and relief of obstruction in the colon at the same time, for wounds in the colon do not unite so readily as those in the small intestine, and are, in addition, exposed to traumatism by the passage of solid fæces. Therefore colotomy should be first done as immediately above the obstruction as seems advisable, and the radical operation left for a later date.

The complete operations we have described as applied to obstruction affecting the small intestine are only possible with a fair chance of success when diagnosis has been made sufficiently early, and surgical treatment has been carried out at once. Patients with acute intestinal obstruction are even worse than they look, and seldom get well after a complete or prolonged operation, however satisfactory, from the operative surgical point of view, if the small intestines are considerably distended. Such an operation, even though it empties the distended intestine, completes the intestinal paresis already commenced, and the patient gets relief after operation only for a few hours.

The operation of choice for such patients is enterostomy. It has been my misfortune to perform this operation, and to find on subsequent post-mortem examination an intestinal opening 12 inches below an impacted gall-stone lying immediately under the anterior abdominal wall; to discover after an immediately successful enterostomy that death on the ninth day was due to gangrene of a loop of bowel ensnared by a string-like adhesion easily accessible, and to see curdled milk oozing through the fistulous opening soon after it had been swallowed, showing that the incision had been made so high in the jejunum that death from starvation must shortly be expected. Against these disappointing results, which a more thorough surgical procedure could have averted only by the certain sacrifice of the patient's life, must be placed the cases in which enterostomy has afforded another chance of life, which the more complete operation did not. A second operation is generally required after enterostomy, but it is not a unique experience to find the symptoms permanently relieved, and to see the patient completely cured, with spontaneous healing of the fæcal fistula.

Enterostomy may generally be regarded as a protest against delay, but until early operation becomes more frequent it holds a useful place in the treatment of intestinal obstruction.

The operation is best done by an oblique incision from above downwards in the direction of the fibres of the external oblique muscle about three inches long, commencing in the right side at the centre of the back of the ileo-costal space and extending as far forward as needful. The cæcum, if distended, appears in the wound, is drawn outwards and opened towards the posterior extremity of the wound, after this has been packed thoroughly with protective gauze. A Paul's tube is then tied into the bowel, the ligature and the junction between bowel and tube are wrapped in iodoform gauze, and the parietes are sutured with silk-worm gut above and below, the two sutures nearest the intestinal opening coming through the outer coats of and fixing the gut.

If the cæcum is not distended the nearest coil of tense small intestine is drawn out and pulled towards the back, opened, and similarly treated.

The advantages of this incision over one in the middle line are that it exposes a favourable site for pathological conditions, the neighbourhood of the ileo-cæcal valve; that if the large intestine be at fault, typhlotomy can readily be done; that there is less difficulty in retaining the intestines in the abdomen with this than with a median incision; that if the small intestine has to be opened, it is here that the ileum, not the jejunum, is likely to be found; and that by keeping the intestinal opening posterior there is less risk of the entrance into the abdomen of its septic contents. This operation can be done with local anæsthesia alone, but it seems better to combine the use of a local with a general anæsthetic. If the skin be frozen with chloride of ethyl, a skilled chloroformist with very few drops of chloroform can dull consciousness enough to render the patient comfortable and incapable of feeling pain or being fearful.

If the fæcal fistula left by this operation shows no tendency to close spontaneously, the abdomen should be opened in the middle line during the third week or thereabouts for the purpose of treating the cause of the intestinal obstruction, and at the same time or later, as seems best, the enterostomy opening can be closed by a plastic operation.

After-treatment of Operation Cases.—The first indications in the after-treatment are to relieve shock and procure rest. To treat shock the patient should be heated in bed by warm bottles and blankets all round, and an enema of two oz. of beef-tea, two oz. of milk, and half oz. of whisky should be administered. His face and

neck and hands should be rubbed with a warm towel and one-twentieth of a grain of strychnine given hypodermically. If recovery does not soon manifest itself, one pint of normal sterile saline should be slowly injected under the pectoral muscle on each side, with strict precautions against septic contamination. For the purpose of procuring rest a single dose of morphia, one-third of a grain hypodermically, is ordered as soon as the patient begins to complain of pain or to get restless. In twelve hours five administrations of calomel in one-grain hourly doses should be commenced unless flatus has been freely passed, when nothing should be given. After five doses of calomel, if flatus still refuses to come away, the insertion of a rectal tube may be tried. If this fail, a turpentine and soap-and-water enema will assist it, and this may be repeated every four hours. Opium should not be given after the first dose unless the patient is obviously dying.

In the matter of feeding, my rule is not to allow more than the smallest quantity of hot fluid, usually a teaspoonful of hot water with a few drops of brandy in it, if required, until flatus has been passed, then to administer small and gradually increasing quantities of milk and barley-water. As soon as free evacuation of the bowels has occurred more solid food may be partaken of.

Intimitis.—Inflammation of the intima or the lining membrane of a blood-vessel. *See* Arteries, Diseases of.

Intolerance.—Undue susceptibility to the effects of a drug or method of treatment; inability to resist such effects.

Intoxication. *See* Alcoholism; Septicæmia; Toxicology.

Intra-.—In compound words *intra-* signifies *within*, *e.g.* intra-abdominal, intracapsular, intra-articular, intracranial, intralaryngeal, intraocular, intraorbital, intraperitoneal, intrathoracic, intra-uterine, intravenous, and intravesical.

Introitus.—The opening of a canal or cavity by which it communicates with the exterior, *e.g.* introitus vaginæ.

Introspection.—In insanity, morbid introspection, or "an unnatural dwelling on and inquiring into one's own acts," is a common symptom. *See* Insanity, Nature and Symptoms.

Intubation. *See also* Diphtheria (*Treatment*); Larynx, Diseases of. The operation of intubation or tubage of the larynx consists in the introduction through the mouth of a metal tube provided with a collar which, when the tube is in position, lies upon the vocal cords. The tubes are of different sizes, and are graduated according to the age of the patient. The introduction is effected by means of an *introducer*, an instrument consisting of a long curved stem and handle, and provided at the end with a screw or catch to which is fixed the jointed metal guide on which each tube fits loosely. A gag having been inserted in the left side of the mouth, the patient may be held in a sitting posture on a nurse's knee, or may be placed lying on the back with the limbs firmly controlled. The operator introduces the left forefinger, and passes it behind the epiglottis. As soon as he feels the opening of the glottis he introduces the tube alongside his finger, and lifting the handle of the introducer, he brings the point of the tube under the tip of the finger and into the opening of the glottis. Then by means of a mechanism which differs in the different instruments he pushes the tube off the guide and leaves it lying in position in the larynx.

As the operation is one of considerable difficulty, and there is always a chance of the tube slipping into the œsophagus, it is absolutely necessary that a loop of thread be passed through a hole in the collar of the tube, so that if the tube is not in position, it can be at once withdrawn. It is usual after the operation is completed to remove this string by pulling on one end, but some prefer to leave it, and fix it by sticking plaster to the outside of the child's cheek. The advantage of this method is that should the tube be suddenly blocked by a piece of membrane, a nurse can pull it out at once, instead of having to wait till the surgeon uses the extractor provided with every set of instruments. The patient readily accustoms himself, moreover, to the presence of the string in the mouth. It may be added, that if the string is to be left in permanently, wire banjo string is most suitable as not being so easily bitten through.

If the obstruction for which the operation has been performed is at, or in the immediate neighbourhood of the glottis, the introduction of the tube relieves the symptoms at once. There is usually a certain difficulty in swallowing at first, but in the hands of careful nurses, if the patient is fed sufficiently slowly, it is easy to supply an adequate amount of nourishment. Soft solids, jelly, baked custard pudding, and so on, are taken better than fluids. Nasal feeding, if necessary, can be resorted to.

If there are no marked signs of irritation and no aggravation of the temperature, the tube may be left in from two to three days, when it should be taken out and cleaned. Quite frequently it is unnecessary to replace it. In any case it should not be reintroduced unless the state of the patient absolutely requires it.

Advantages.—The advantages of this operation are that it may often prevent the necessity of a cutting operation, so dreaded by parents, and that it, in the case of diphtheria, gives time

for antitoxin to stop the spread of the disease without exposing the patient to a tracheotomy. The *disadvantages* are unfortunately more numerous. In the first place, it cannot be said to give rest to the affected part. Secondly, the tube may push down membrane before it, and so inoculate the larynx lower down. Thirdly, if the membrane is loose, it may be even rolled up in a ball by the introduction of the tube, and absolutely plug the larynx below the point of the instrument. And although these objections which are largely theoretical may be discounted, it is impossible to dispute that the operation is totally unsuited for general practice. In the first place, constant practice is required if the operator is to give his patient every chance. Secondly, a nurse cannot replace a coughed up tube as she can that of a tracheotomy, and as this accident frequently happens, unless a doctor can be always on the spot the patient's life is not safe.

In infectious hospitals, however, where a large amount of diphtheritic croup is treated, and where there are resident medical men, the introduction of antitoxin has given a new lease of life to intubation as an operation. It is always worth trying in the first place, and if it does not relieve the patient, tracheotomy should be done at once without waiting too long. It is quite unusual nowadays to see membrane spread after antitoxin has been given in adequate doses, and therefore the objections regarding the inoculation of healthy parts of the larynx no longer hold. There is no reason why an operation which is so popular in America and France should not give good results in hospital practice in this country.[1]

Intussusception. *See* Intestines, Surgical Affections of (*Intestinal Obstruction, Causes*); Gastro-Intestinal Disorders of Infancy (*Constipation*); Peritoneum, Acute Peritonitis, General (*Etiology*).

[1] The usual instruments used are those of O'Dwyer. They have been modified by Colin, whose tubes are shorter and whose introducer is more easily kept clean.

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Printed by R. & R. CLARK, LIMITED, *Edinburgh.*